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Archives of Neurology and Psychiatry

VOLUME 14 DECEMBER, 1925 NUMBER 6

GENERAL SIGNIFICANCE OF TAY-SACHS DISEASE

PROF. CHARLES SCHAFFER

BUDAPEST, HUNGARY

Although both the histopathologic and the clinical details of Tay-Sachs disease have been treated minutely, the general significance of this heredofamilial disease apparently is not properly recognized. I welcome the opportunity to publish a special paper on this in the United States, as America is the classical realm of investigations of this kind. It is sufficient to refer to the fundamental and excellent publications of Dr. B. Sachs, New York, and to the communications of Hirsch, Spiller, Strauss, Globus and other American authors.

I shall discuss: (1) the general points of view on the histopathology of Tay-Sachs disease; (2) the explanation of Tay-Sachs disease and of heredofamilial system disease by conclusions drawn from the histopathologic findings. I have dealt with both in my previous works, and I come back to these questions because the histopathology of Tay-Sachs disease has been enriched by interesting new contributions, and because the general conclusions which I drew concerning the histopathology of hereditary diseases of the nervous system have not yet found their way into general knowledge in accord with their importance.

The general feature in the histopathology of Tay-Sachs disease is the swelling of the neurons. The nerve cells may swell as a whole or only in part, as in the dendrites or axons, which may be swollen like a balloon in a localized area (Fig. 1). The swelling is extreme in most cases, and its extension to the dendrites and the cell bodies imparts to it a special character which does not occur in exogenous, experimental swellings (Nissl's primary irritation). However, it is not only the degree, that is, the intensity of the swelling in individual nerve cells, but also the distribution, that is, the extent, which is a distinctive feature of the histopathologic picture of the disease which is not found in the exogenous diseases, for the ubiquitous swelling of the cells which develops from the cortex down to the conus medullaris is found in no other process.

It is an important fact that this swelling also involves the ectodermal multipolar glia cells, giving rise to monster or irritation glia cells. It is characteristic of Tay-Sachs disease that the swelling affects the ectoder-

mal elements while the mesodermal elements (membranes and blood vessels) remain unaffected.

Examining the swellings more closely, we easily recognize that of the differentiated and undifferentiated elements of the cell body, only the structureless cytoplasmic element—the hyaloplasm—swells. Those nerve cells which present—both when stained to demonstrate fibrils and when stained with hematoxylin-fuchsin preparations—a barred frame-

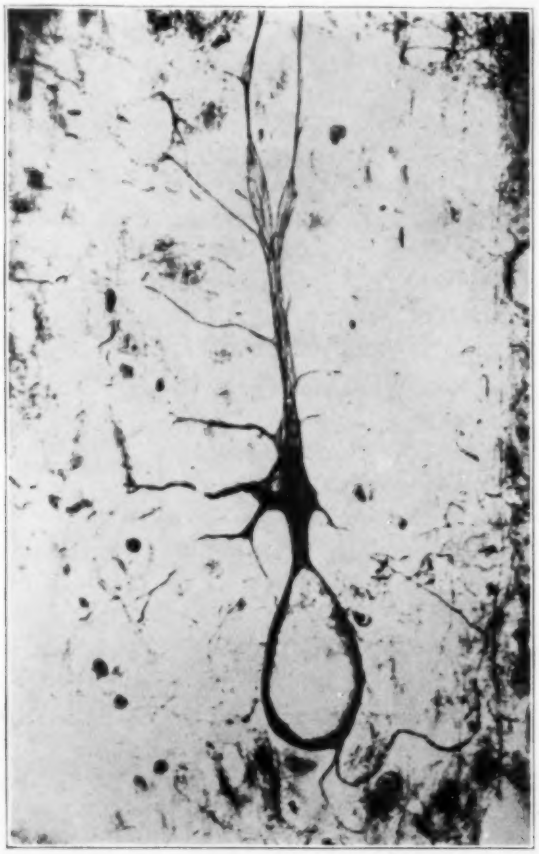

Fig. 1.—Balloon-shaped swelling of basal dendrons of a pyramidal cell of the cerebral cortex in Tay-Sachs disease. The swollen portion shows the superficial fibril shell as a black border. There is detritus in the shell. The apical dendron is moderately swollen as a whole and the fibrils have spread apart.

work, the gaps of which appear empty in fibril preparations, correspond to this condition. On the contrary, in fuchsin preparations, they show a slightly tinged homogeneous substance (hyaloplasm) which as a result of the swelling has filled the gaps. I have called this stage the swelling phase, and it is followed by the precipitation phase, in which

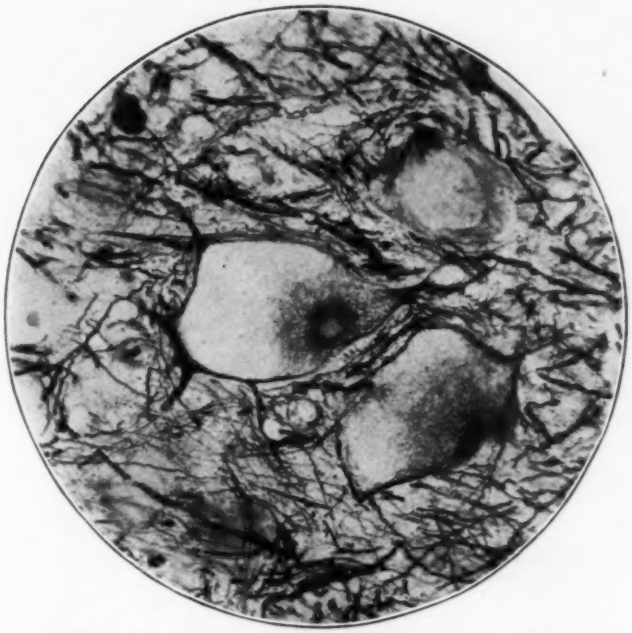

Fig. 2.—Swollen cells of the anterior horn of the spinal cord in Tay-Sachs disease. The fibrils are pressed to the surface forming a fibrillary shell. The internal net is visible only around the nucleus in the form of detritus. Fibril impregnation of Bielschowsky.

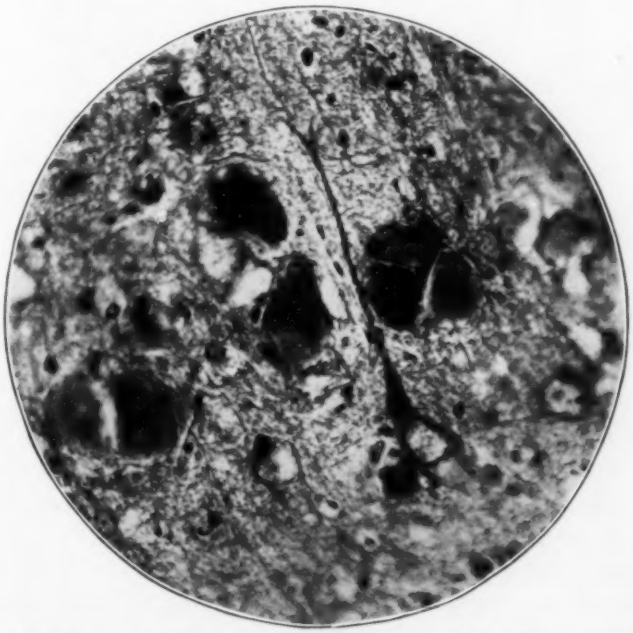

Fig. 3.—Swollen astrocytes (mast of irritation glia) in the pyramidal layer; between these is a swollen nerve cell.

granules appear in the swollen hyaloplasm, followed by round or polygonal bodies, which stain with hematoxylin and which fill the gaps. These gradually developing bodies are prelipoid degeneration products which from their staining properties I have considered similar to lecithin.

In the late phases of familial idiocy (Schob, Walter) this prelipoid degeneration substance appears to be changed to fat (Herxheimer's and osmic acid fat stain), the degeneration granules thus undergoing a gradual fat transformation.

This description shows that the edema is caused not by the accumulation of prelipoid degeneration products, as Bielschowsky

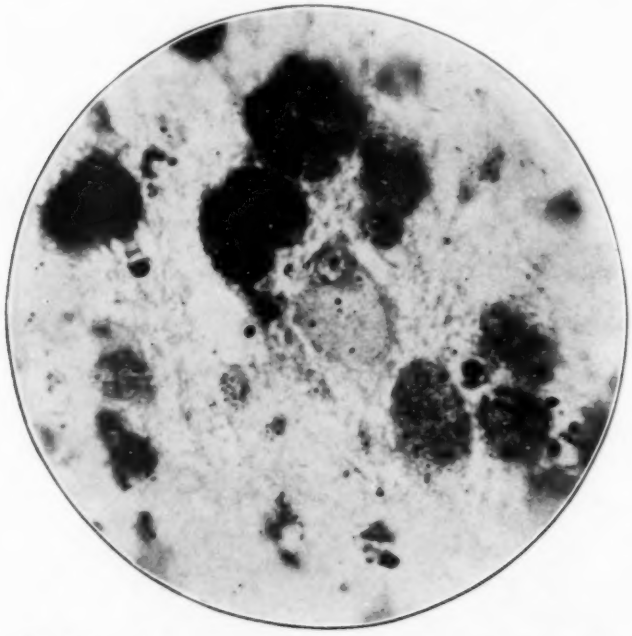

Fig. 4.—Swelling of a nerve cell of the cerebral cortex (in the center of the field). The nucleus is driven toward the apex. In the swollen cytoplasm four or five granules are to be seen as the sign of beginning precipitation. Other nerve cells are to be found in various stages of precipitation. Weigert's myelin sheath stain overstained by fuchsin.

and Spielmeyer suggested, but by the swelling of the structureless hyaloplasm; from this the lecithinoid bodies arise. It seems to be certain that the neuron constituent primarily affected is the hyaloplasm. The heredodegenerative process classically shown in Tay-Sachs disease is localized in the hyaloplasm.

Of course the cytopathologic process does not remain stationary at this stage of the swelling. Even though the subacute course leads quickly to a lethal end and microscopic examination shows swelling

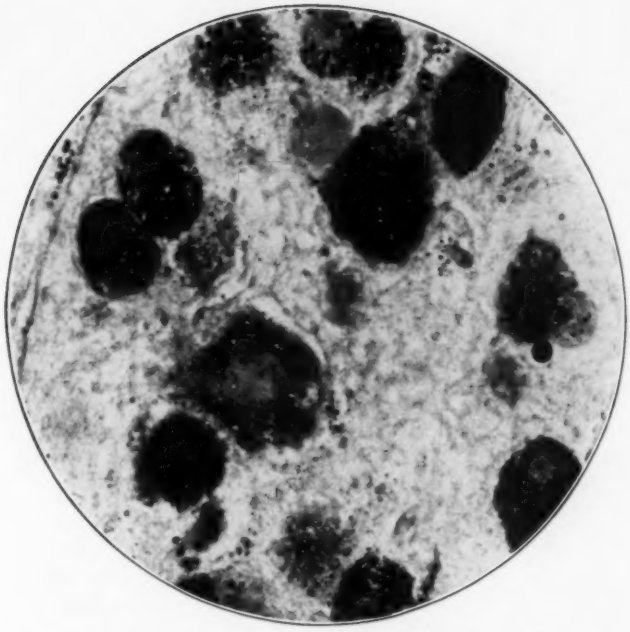

Fig. 5.—Lecithin-like degeneration of the cortical cells in Tay-Sachs disease. Between the nerve cells which are entirely occupied by hematoxylin-eosinophil granules, a swollen nerve cell is to be seen toward the center of the field, its nucleus lying at the side. This illustration presents the development of the condition represented in Figure 4.

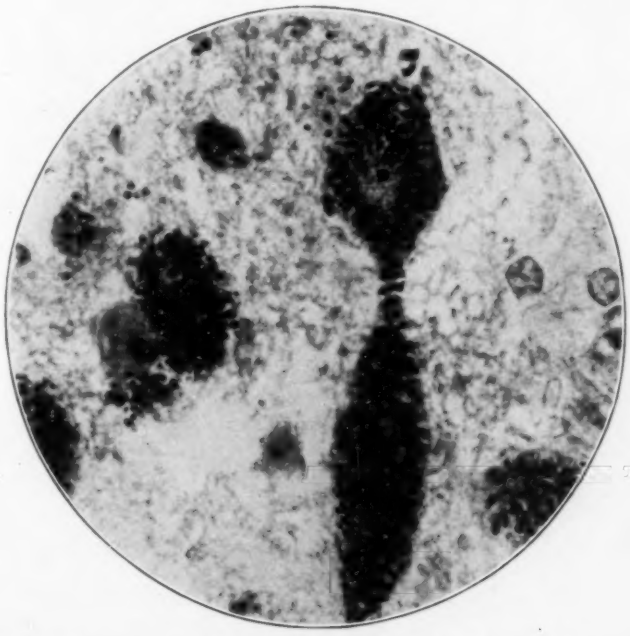

Fig. 6.—Precipitation stage of a pyramidal cell of the cerebral cortex; both the swollen cell body and the expanded swollen basal dendrite are completely filled with lecithin-like degeneration products. On the left of this nerve cell there is a formless swollen nerve cell loaded densely with degeneration products. Next to this cell is an apolar neurophagic glia cell. Weigert and fuchsin stain.

still in the florid stage, yet collapsed nerve cells appear here and there; that is, in fibril preparations one finds nerve cells with a superficial fibril coating which either contain reduced detritus only or are entirely empty. In hematoxylin-fuchsin preparations such nerve cells show only a few lecithin-like bodies which are slightly stained, and it is striking how much they are reduced. This reduction is caused by the neuronophagic

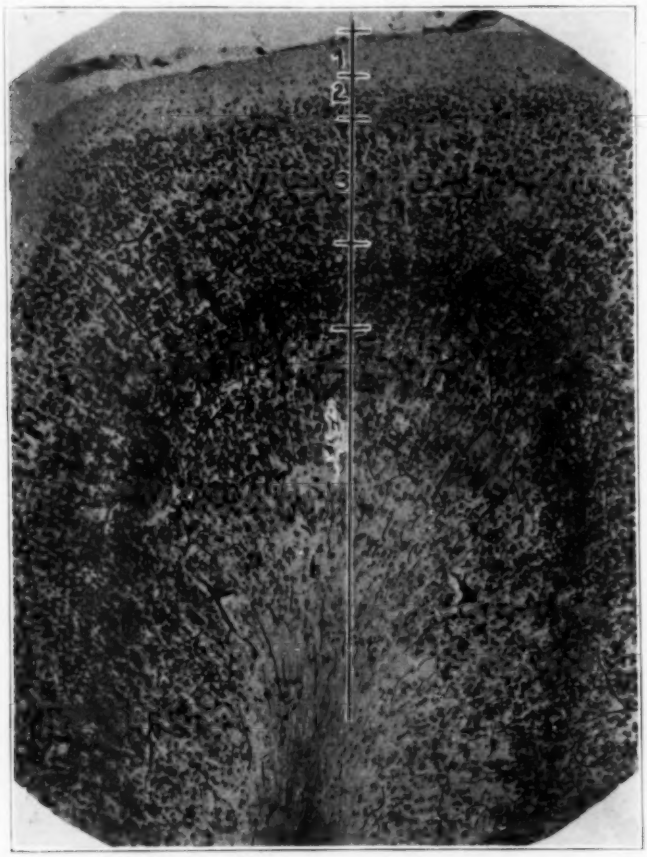

Fig. 7.—A frontal gyrus which as a result of the ubiquitous granular degeneration of the nerve cells presents the laminated (tectonic) structure of the "granular" frontal gyrus. 1 indicates the lamina zonalis; 2, lamina granularis externa; 3, lamina pyramidalis; below is the marked lamina granularis interna, then the lamina multiformis.

action of apolar glia cells. These elements adhere to the swollen nerve cells and take up, evidently through osmosis, one part of the pathologically altered cytoplasm, which within the cell body is quickly transformed to fat. This action is to be seen in the apolar glia cells, which are filled with scarlet droplets. These cells thus become glial granule

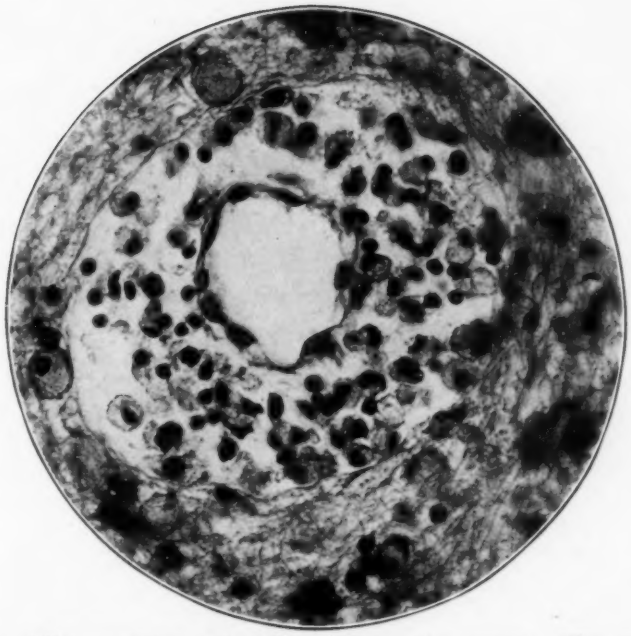

Fig. 8.—Glial granular cell infiltration in the cortex. The enormously dilated perivascular space with numerous cells entirely filled with granules is limited by a distinctly outlined layer (membrana limitans gliae perivascularis), to which adhere giant granular cells. Van Gieson's stain.

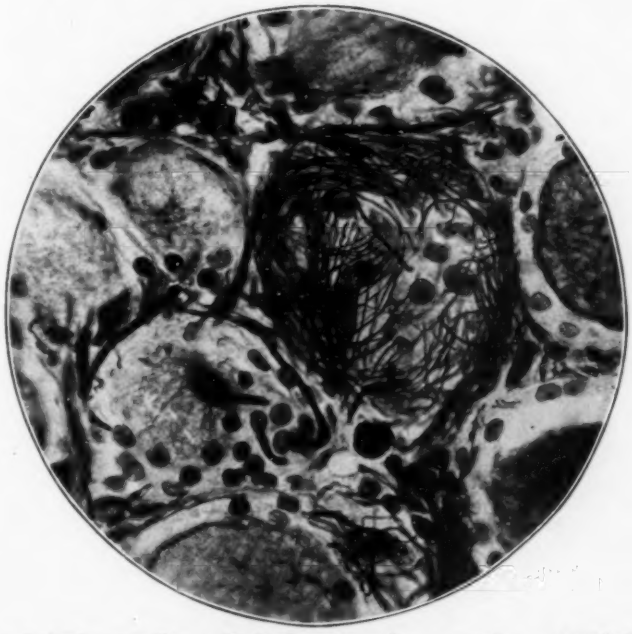

Fig. 9.—Spinal ganglion; fibril impregnation reveals a tangled mass of fibers under the capsule. (Concerning the significance of the latter, see C. Schaffer: Contributions to the Histopathology of the Spinal Ganglions, Arch. f. Psychiat. 67.)

cells. They reach the perivascular lymph spaces by their ameboid motion, and cause a gliogenous granule infiltration. Finally, the spinal ganglia are affected.

The swelling is much less developed in the medullated nerve fibers than in the cells. Striking decomposition of the medullary sheaths is seldom noted, but the symptom of cessation of the myelinization process appears, that is, the systems of Flechsig in which the formation of

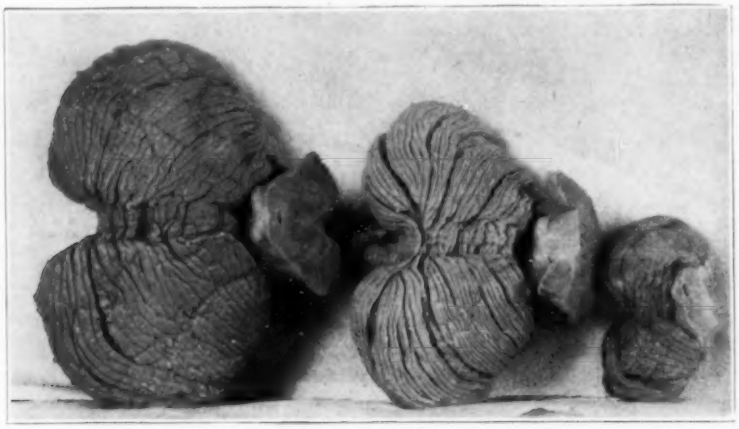

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Fig. 10.—Three cerebellums. The first is that of a normal person; the second that of a deaf and dumb person with ataxia and the third that of an idiot. The second and third show hypoplasia.

Fig. 11.—Cervical segment of a person with Tay-Sachs disease. The lateral pyramidal tracts are faint in contrast to Flechsig's tract. Note also the bilateral notching of the lateral columns in the vicinity of the posterior horns. This is the lateral sulcus, a fetal relic. Wiegert's stain.

medullary sheaths occur late do not reach the medullated stage. Among these are the pyramidal tracts, corticothalmic, corticopontile and ponto-cerebellar connections. In brief, in Tay-Sachs disease the fiber systems present not so much a degeneration as a cessation of myelinization.

CHARACTER OF PATHOLOGIC PROCESS

I wish to call special attention to the following facts:

1. The process is a purely ectodermal one, which leaves the mesodermal elements of the central nervous system entirely unaffected. It is a selective process in which the chief disturbance is an ectodermal decomposition combined with an ectodermal scavenging process which is

Fig. 12.—Twin cells in a spinal ganglion.

effected by glial fat-granule cells. One seldom perceives a glial compensatory reaction in the brain. In the cerebellum, fiber formation sometimes occurs in the neuroglia coat of the molecular layer, and in consequence at the same time a striking hypoplasia of the cerebellum occurs. Since the process is selectively ectodermal and entirely independent of external influences, being an endogenous hereditary malady, it may be a single germ layer disease. This process has nothing in common with the exogenous ectodermotropic processes.

2. The alterations have a systemic character, for the systems in which the medullary sheaths develop at a late stage are arrested in their development of myelin. This interruption of myelinization is bilateral

and shows an embryologic factor, because those systems are attacked which are retarded, that is, the most labile in the course of the evolution of systems which under normal conditions reach maturity the latest.

To this germ layer selectivity and the system selectivity may be added a third, an embryologic, factor. This is seen to best advantage in other chronic heredofamilial nervous diseases, such as the heredospinal and cerebellar forms, in which an underdevelopment—hypoplasia—is always noticeable in one segment, that is, in the cerebellum or in the spinal cord.

Three Features Characteristic of Various Heredofamilial Nervous Diseases

Heredofamial Diseases	Germ Layer Affected	System Affected	Segment Affected
1. Hereditary pyramidal tract affection (Strümpell)	Only the elements of the ectoderm diseased; no trace of mesenchymal affection	Pyramidal tract together with motor cortex (Betz), sometimes also tract of Goll and hence a combined system disease	Telencephalon plus spinal cord, single segmentary affection or combination
2. Hereditary extrapyramidal disease			
Chorea herpes simplex	Only the elements of the ectoderm diseased; no trace of mesenchymal affection	Microcellular part of the striatum; single system disease	Telencephalon (striatum); single segmentary affection
Chorea of Huntington	Only the elements of the ectoderm diseased; no trace of mesenchymal affection	Microcellular part of the striatum, third and sixth layer of the frontal cortex and ganglia, hence a combined system disease	Telencephalon (striatum plus cortex of the cerebrum) single segment
3. Heredocerebellar disease	Only the elements of the ectoderm diseased; no trace of mesenchymal affection	Rhomboidal brain system: cerebellar and paracerebellar, i. e., a combined system disease	Rhomboidal brain, i. e., single segmentary
4. Friedreich's disease	Only the elements of the ectoderm diseased; no trace of mesenchymal affection	Spinal cord systems: Goll, Flechsig, hence a combined system disease	Spinal cord, that is, single segmentary
5. Familial spinal amyotrophia	Only the elements of the ectoderm diseased; no trace of mesenchymal affection	Anterior horn system, that is, a single system disease	Spinal cord, single segmentary
6. Tay-Sachs or infantile family idiocy	Only the elements of the ectoderm diseased; no trace of mesenchymal affection	Ubiquitous nerve cell degeneration, hence all centers attacked; systems which develop myelin sheaths at a late stage are arrested	All segments, hence all segmentary disease

In such cases the process is confined to a segment, and a segment selection manifests itself. In the course of Tay-Sachs disease, the segment selection is expressed only by participation of the cerebellum resulting in a strikingly small cerebellum. Sachs, and later I, pointed out the spreading of the Sylvian fissure as a sign of the hypoplasia of the cerebrum. Regarding the spinal cord, I have often seen a lateral sulcus of the lateral column. As a microscopic sign, I have found twin cells in the spinal ganglia. From these data we gather that in Tay-Sachs disease

the central nervous system, that is, the brain, spinal cord and the spinal ganglia, all present signs of defective development representing underdevelopment of the entire central nervous system. The ubiquitous nerve cell process corresponds to this finding. Tay-Sachs disease is an all-segmentary process as compared with the familial spinal amyotrophy either of Friedreich or of Marie, which are affections of a single segment. Further, there exist combined segmentary processes in which Friedreich's disease plus Marie's disease or amaurotic idiocy plus Marie's disease, etc., occur in combination.

Germ layer selectivity, system selectivity and segment selectivity characterize in a specific way a group of diseases of the central nervous system which have certain special clinicobiologic marks, namely: heredity, familiality, consanguinity and race selection. The heredofamilial nervous diseases, being developed on a hereditary biologic basis, have both clinically and anatomically a special character. They are distinguished fundamentally from the nervous diseases of exogenous origin, which as a rule do not affect selectively the elements of one single germ layer and do not spare certain systems and segments. It happens, however, that certain poisons manifest a particular affinity for ectodermal elements and cause a degeneration (diphtheria toxin, etc.) suggesting an ectodermotropic action. These conditions are not, however, combined with a systemic and segmental attack. At the same time there are often signs of mesodermal reaction (inflammation), the traces of which prove the exogenous character of the process.

SUMMARY

Thus we see that the systemic heredofamilial nervous diseases from the histopathologic standpoint are marked by three features: germ layer selectivity, system selectivity and segment selectivity. These three fixed factors form the factor triad which I have pointed out, and they attest an embryologically determined substratum. They are genotypic or idiotypic diseases, and so they can be explained only by the laws of embryology. In general, they are ectodermogenous diseases rather than ectodermotropic diseases. The term ectodermogenia means genotypia; ectodermotropia is synonymous with paratypia. As an example: Take as a phenotype, first, the spastic paralysis of Strümpell or hereditary spastic paralysis, and second, the syphilitic spinal paralysis of Erb; the first corresponds to idiotypic familial diseases, the second to paratypic nonfamilial diseases. For the first the factor triad is in evidence (see table); in the second there is an exogenous factor (syphilis) which affects a systemic group (lateral tracts), and in this way imitates true familial spastic paralysis.

In the table I have arranged some of the most important heredofamilial nervous diseases in relation to the factor triad.

BREECH DELIVERY IN ITS POSSIBLE RELATIONS TO INJURY OF THE SPINAL CORD

WITH SPECIAL REFERENCE TO INFANTILE PARAPLEGIA *

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BALTIMORE

Birth injuries of the spinal cord resulting from breech extraction have been known to pathologists for many years. Probably the first case of this nature was reported by Parrot [1] in 1870. Since then occasional cases of cord injury from breech extraction have been found in almost every considerable series of necropsies on the stillborn. Good descriptions of the pathologic findings may be found in articles by Spencer,[2] Warwick [3] and Stoltzenberg.[4] Among obstetricians, injury to the cervical cord is recognized as a rare cause of infant mortality; but the standard textbooks of neurology and of pediatrics do not mention obstetrical injuries of the cord as a cause of spinal paraplegia. Apparently, it is generally believed that the lesion is always fatal within a few days after birth.

Scattered reports of patients who survived birth injuries to the cord for longer periods, however, have appeared from time to time. Thus, Beevor,[5] in 1902, described a case in a child who lived fifteen weeks, and Burr,[6] in 1920, reported two more cases in children, each of whom lived five months. The same year Kooy [7] gave the clinical and pathologic findings in the case of a child who lived eight years. Three similar cases have appeared in the German literature, by Handwerk,[8] Gött [9] and

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* From the Medical Clinic, Johns Hopkins University Medical Department.

1. Parrot: Note sur an cas de rupture de la moelle, chez un nouveau-né par suite de manoevres pendent l'accouchement, L'Union méd. **11**:137, 1870.

2. Spencer, H. R.: Visceral Hemorrhages in Still Born Children, Tr. Obst. Soc. London **33**:203, 1892.

3. Warwick, W.: Necropsy Findings in New-Born Infants, Am. J. Dis. Child. **21**:488 (May) 1921.

4. Stoltzenberg: Zerreissungen der intervertebralen Gelenkkapseln der Halswirbelsauüle, eine typische Gebutsverletzung, Berl. klin Wchnschr. **37**:1741, 1911.

5. Beevor, C. E.: A Case of Hemorrhage into the Spinal Cord at Birth, Brain **25**:85, 1902.

6. Burr, C. W.: Hemorrhage into the Spinal Cord at Birth, Am. J. Dis. Child. **19**:473 (June) 1920.

7. Kooy, F. H.: Rupture of the Spinal Cord in Dystocia, J. Nerv. & Ment. Dis. **52**:1 (July) 1920.

8. Handwerk: Zur pathologischen Anatomie der durch Dystokie entstandenen Rückenmarksläsion, Virchow's Arch. f. path. Anat. **154**:169, 1901.

9. Gött: Ueber einen seltenen Lähmungstypus nach Geburtstrauma, Jahrb. f. Kinderh. **69**:422, 1909.

Lawatschek.[10]. Nevertheless, these articles seem to have attracted little attention, and it is probable that many cases passed unrecognized. In 1923, an excellent paper appeared by Crothers,[11] in which he described five cases of spinal paraplegia following breech extraction and clearly outlined the clinical picture. He claimed that the condition was not infrequent, especially since the recent widespread adoption of version as a routine procedure, and that it was generally unrecognized because the symptoms of complete transection of the spinal cord in children had been imperfectly understood. Crothers also suggested that herniation of the medulla into the foramen magnum might result from traction on the cord, and that this might contribute to the mortality of breech extraction.

No additional cases have appeared since Crother's paper, and, therefore, it seems desirable to report six more cases of birth injury of the spinal cord, and to call attention again to one of the dangers of breech extraction.

REPORT OF CASES

CASE 1.—*Breech extraction, rupture of the dura and laceration of the cord at the eighth cervical; flaccid paraplegia with abolition of tendon reflexes; death on third day; necropsy.*

Baby B., full term child of healthy parents, was born Dec. 1, 1924, in the Church Home and Infirmary. The breech presented, and, it is said, delivery was accomplished without forcible traction. The weight at birth was 9 pounds (4.1 kg.). The child did not cry well, and was very cyanotic; but there were no convulsions or bulging of the fontanels. The legs were flaccid and did not move. No loss of motility was noticed in the arms. Lumbar punctures on the second day revealed blood stained fluid. Death occurred on the third day. The clinical diagnosis was meningeal hemorrhage.

Necropsy revealed rupture of the dura and leptomeninges at the dural exit of the eighth cervical roots. The ends of the dura were separated almost 2 cm. The cord was stretched and softened at this level, and surrounded by blood clot. When sectioned it was found to be infiltrated with blood. The changes in the cord were confined to the eighth cervical and first thoracic segments. A thin layer of clotted blood outside the dura extended up and down for several segments above and below the lesion. No intracranial lesions were found except a little blood in the subarachnoid spaces. The tentorium was intact. There was no fracture or dislocation of the vertebrae, but minute hemorrhages were seen beneath the posterior common ligament and in the intervertebral disks of the seventh cervical and the first two thoracic vertebrae. The spinal ligaments were intact.

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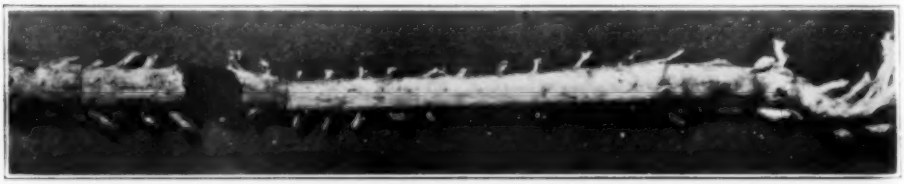
10. Lawatschek: Ein Fall von Hämatomyelie mit kompletter Leitungsunterbrechung bei einen Neugeborenen mit viermonatlicher Lebensdauer, Arch. f. Kinderh. **56**:1, 1911.

11. Crothers, B.: Injury of the Spinal Cord in Breech Extraction as an Important Cause of Foetal Death and Paraplegia in Childhood, Am. J. M. Sc. **165**:94 (Jan.) 1923.

CASE 2.—*Version and extraction; complete physiologic transection of cord in upper thoracic region; active tendon reflexes and flexion reflex; no improvement in three months.*

Evelyn S., colored, third child of healthy parents, was examined at the age of 3 months in the Harriet Lane Dispensary. The history was as follows: Labor was long and difficult. Forceps were tried unsuccessfully, and finally podalic version was performed. The baby was deeply asphyxiated and resuscitated with difficulty. Weight at birth was 11 pounds (5 kg.). No convulsions were observed; but the child never moved her legs, and her cries were feeble. A diagnosis of amyotonia congenita had been made before the child was brought to the dispensary.

Examination revealed a well nourished and well developed child. Mentality was apparently unimpaired. The cranial nerves were normal. The arms moved freely, and when the body was supported, the head was well sustained. No spontaneous movements of the legs were observed. The muscles of the legs were of good bulk, but slightly soft, and passive movements showed some diminution of tone. The abdomen was soft and bulging. Respiration was entirely diaphragmatic, and there was definite retraction of the lower ribs during inspiration. When the child was placed in a sitting position, her back buckled, and she fell forward. Urine was passed frequently in small



Spinal cord in Case 1, showing rupture of meninges and laceration of the cord.

amounts. The bladder was not distended. The bowels were persistently constipated. The child did not respond to pinching of the skin below the nipple line. The tendon reflexes in the legs were increased, and a bilateral ankle clonus was obtained. Plantar stimulation caused flexion of the toes and other joints of the lower extremity. This response was usually bilateral. The abdominal reflexes were not obtained. There were no trophic changes in the skin. No abnormality of the spine could be made out.

Examination of the chest revealed signs of diffuse bronchitis. After several days in the hospital, the child developed pneumonia and was taken home despite the remonstrances of the ward physician. Two weeks later the child died. Necropsy was not performed.

CASE 3.—*Breech extraction; complete physiologic transection of cord in upper thoracic region; active tendon reflexes, and flexion reflex; no improvement after one year and four months.*

Sarah H., fifth child of normal parents, was examined in the Harriet Lane Dispensary at the age of 1 year and 4 months. The following history was obtained: The breech presented, and the child was extracted manually after eighteen hours. Cyanosis was marked and resuscitation was difficult, but no convulsions or twitchings were seen. Paralysis of the legs was noted soon after birth, and this had persisted. Constipation had always been troublesome, but the child had been healthy, and development had seemed normal.

Examination revealed a well nourished and well developed child with a mucopurulent nasal discharge. There was no apparent defect in mentality. The arms moved freely, and no disturbances of the cranial nerves could be detected. The legs were flaccid, and no spontaneous movements were observed, but no muscular atrophy could be demonstrated. Respiration was diaphragmatic, and during inspiration the lower ribs were retracted. The upper half of the chest seemed to move normally. The child could not sit up, but could sustain her head when her body was supported. The bowels were constipated, and there was constant dribbling of urine. Pinching of the skin below the nipple line caused no sign of pain. The tendon reflexes in the legs were increased, and bilateral ankle clonus could be elicited. Plantar stimulation produced extension of the toes and flexion at all the other joints of the lower extremity. This response was usually unilateral, but was sometimes bilateral. The abdominal reflexes were not obtained. The skin showed no trophic changes. No abnormalities of the spine could be detected, and a roentgenogram was negative.

Severe rhinopharyngitis was present when the child was first seen, and a few days later the signs of bronchitis were found. The patient was discharged unimproved.

CASE 4.—*Version and extraction; complete physiologic transection of cord in upper thoracic region; tendon reflexes increased, "mass reflex"; no improvement after one year and six months.*

Jeanne L., first child of healthy parents, was examined in the Harriet Lane Dispensary at the age of 1 year and 6 months. The history was as follows: Labor was prolonged for twenty-four hours, and was finally terminated by version after an unsuccessful attempt to apply forceps. Weight at birth was 8 pounds (3.6 kg.). No convulsions occurred, but cyanosis was marked. The child cried at once, but very feebly, and nursed with difficulty. It was noted in the first few days that the legs did not move, and the arms moved feebly. The whole body was "limp as a rag." On the fifth day, retention of urine occurred, and the bladder was emptied twice with a catheter. Cystitis soon developed, and since then there has been constant dribbling of urine. At ten months, the baby could hold her head up and move her arms freely. Her legs were no longer limp but rather stiff. A diagnosis of amyotonia congenita had been made, and a good prognosis had been given.

Examination revealed a bright, alert child of normal development for her age. The cranial nerves were normal. No disturbance of motility in the arms was apparent, but the legs were paralyzed in extension and slightly spastic on passive movement. The abdomen was soft and flabby. Respiration was diaphragmatic, and no excursion of the costal margins could be seen during inspiration, but there was no retraction of the lower ribs. The child could not sit up because of weakness of the lower extensors of the spine, but she could hold her head up normally. Marked distention of the bladder was present, and the bowels were constipated. Pricking with a pin was unnoticed below the level of the second rib. The tendon reflexes were increased in the legs, and bilateral ankle clonus was elicited. Plantar stimulation caused extension of the toes and sometimes a general movement of flexion of all the joints and discharge of a few drops of urine. The skin was soft and smooth, except for slight redness and scaliness on the buttocks, where it was subject to irritation by the urine. The spine was flexible, and no irregularity of contour could be

found. A poor prognosis was given, although the predominance of tone in the extensor muscles would suggest that there was not a complete anatomic transection of the cord.

CASE 5.—*Version and extraction; almost complete interruption of conduction in the spinal cord at upper thoracic level; active tendon reflexes and flexion reflex; little improvement in six months.*

Frank H., first child, born at full term of healthy parents, was examined at the age of 6 months in the Harriet Lane Dispensary. There was a history of labor lasting twenty-one hours, which was finally terminated by version. Great force was employed, and traction was made by the obstetrician and an assistant pulling together. There was marked cyanosis at birth, but no convulsions and no bulging of the fontanels. Soon after birth it was noted that the child did not move his legs, and this condition had persisted with little improvement. Otherwise development had seemed normal. Constipation had been troublesome.

On examination the child seemed bright, alert and well developed for his age. The cranial nerves were normal. The arms moved freely, and the grips were strong. The child could hold his head up well, but could not sit up because of weakness of the lower extensors of the spine. Except for a feeble movement of flexion at the left hip when the child tried to crawl, no spontaneous movements of the legs were observed. Passive movements of the legs showed slightly diminished tone, but the muscles were of fairly good bulk and only slightly soft. The abdomen was bulging, and the abdominal muscles seemed flabby. An inguinal hernia was present on the left. Respiration was diaphragmatic, and the lower ribs did not show any definite excursion on inspiration. There was a frequent discharge of small quantities of urine. Almost complete loss of cutaneous sensibility was found over the lower part of the body, but no definite level was demonstrated. The tendon reflexes were active in the legs, and ankle clonus could be elicited bilaterally. Plantar stimulation caused dorsal movement of the toes and flexion of all the other joints of the leg. This movement was strictly unilateral. The abdominal reflexes were not obtained. There were no trophic changes in the skin. The spine was normal.

CASE 6.—*Version and extraction; complete physiologic transection of the spinal cord at the upper thoracic level; active tendon reflexes and "mass reflex"; no change after seven weeks.*

Roland S., first child of healthy parents, was examined in the Harriet Lane Dispensary at the age of 7 weeks. The parents gave the following history: Labor lasted two days, and at last was terminated by version and manual extraction after forceps were tried repeatedly without success. During extraction, it is said, something was felt to "give way." The baby was large, and after birth was deeply asphyxiated. He had never moved his legs, but in other respects had seemed a normal child.

Examination revealed a well developed child. General nutrition was good. The cranial nerves were normal, and the arms moved actively. The legs were slightly flexed and perhaps somewhat flaccid on passive movement, but no atrophy could be made out. No spontaneous movements of the legs were seen. The flanks bulged, and the abdominal walls were relaxed. During inspiration there was retraction of the lower ribs. The child could sustain his head, but could not sit up because of weakness of the extensors of the spine. Urine was passed almost constantly in small amounts, and there was marked constipation. Cutaneous sensibility was diminished or lost on the legs and the lower part

of the trunk, but no definite level was found. Bilateral ankle clonus could be elicited, and the tendon reflexes were increased in the legs. Plantar stimulation caused extension of the toes, flexion of both legs and a spurt of urine. Sometimes, instead of being flexed, the contralateral leg was extended. The abdominal reflexes were not obtained. No trophic changes in the skin were noted. Clinically the spine seemed normal, and a roentgenogram showed no lesion.

DISCUSSION

Five of the foregoing cases were seen within the course of two years, and it is evident that paraplegia from breech extraction is not as unusual as has been supposed. It seems probable that most cases are unrecognized. In fact, only one case of the six described in this article had been correctly diagnosed before examination at the hospital. A history of dystocia is almost invariably obtained. In this series labor was prolonged in five cases, and in two cases it is certain that excessive force was applied during extraction. In four cases version was performed to terminate difficult labor. The other two cases were spontaneous breech presentations.

There was no clinical evidence of injury to the spinal column in any case, and roentgenograms in two cases were negative. In the fatal case nothing was found to indicate injury to the spine except a little blood under the posterior common ligament and in the intervertebral disks. Kooy, Burr and Crothers failed to find any sign of fracture or dislocation of the spine, and it is evident that stretching of the cord, and not crushing, is the essential cause of the lesion.

In the first case the lesion was limited to the eighth cervical and first thoracic segments. The clinical evidence in the other five cases indicated that the lesion extended up to the upper thoracic segments in three, and to the middle thoracic segments in the other two cases. The exact upper level was difficult to determine because of the impossibility of a satisfactory examination of sensibility in infants and of the difficulty of detecting muscular atrophy. It was even more difficult to estimate the extent of the lesion downward in the spinal cord. The relaxation of the abdominal muscles would suggest that the lower thoracic cord was involved, but the thigh muscles were also flabby and atonic, although the active knee reflexes proved that the lumbar cord was intact. In Burr's two cases, the lesion involved the lower part of the cervical enlargement, but the cases reported by Kooy, Beevar and Crothers all showed thoracic lesions. In Beevor's case the whole thoracic cord was destroyed by hemorrhage, but the other cases that came to necropsy showed transverse lesions limited to a few segments. We may conclude that in those who survive the immediate effects of the injury, the lesion is usually in the upper part of the thoracic cord. High cervical lesions are, of course, immediately fatal.

The publication in 1917 of Riddoch's paper [12] on war wounds of the spinal cord has led to a better understanding of the symptomatology of complete transections of the cord; and we know that the reflex activities of the severed segment may be strikingly evident or almost wholly in abeyance, and we may expect a series of clinical pictures varying between these two extremes. At one end of the series we have the familiar picture described by Bastian,[13] with complete loss of tendon reflexes, wasting of the muscles, retention of urine and rapid development of bed sores and other trophic lesions. At the other end of the series we may have, in exceptional cases, an extraordinary activity of the lower segments, with active tendon reflexes, reflex flexion of both legs, automatic emptying of the bladder and almost complete absence of muscular atrophy and trophic disturbances. In some cases stimultation of the sole of the foot will cause violent flexor spasm of the legs, discharge of urine and sweating of the skin below the lesion. This is termed the "mass reflex." These reflexes do not appear at once, but develop gradually only after several weeks during which the muscles are flaccid and there is retention of urine. Bed sores, cystitis, or any infection that depresses the patient's general condition may prevent the development of reflexes or abolish them after they have become established. Riddoch, therefore, distinguished three stages: the stage of "spinal shock" when the reflexes are absent; the stage of reflex activity, which may last indefinitely if there are no complications; and the stage of abolition of the reflexes. In civil life, however, the vast majority of adults with complete transections of the spinal cord show few signs of spinal automatism, and cases showing the "mass reflex" are unusual. Infections and intoxications are almost unavoidable, and the average case closely resembles Bastian's description.

It is well known that after experimental section of the spinal cord in young animals the reflexes are more active and appear after a shorter latent period than those of older animals of the same species; and Crothers has suggested that the reflexes of the severed cord may be more active in children than in adults. No reflex activities could be expected in Case 1, because the child died on the third day while still in the stage of spinal shock. The other five cases of my series all showed active tendon reflexes and the flexion reflex, and two cases showed the complete "mass reflex." The tendon reflexes were just as active in Cases 2, 3, 4 and 6, in which there was complete interruption of conduction, as they were in Case 5 in which paralysis was not complete.

12. Riddoch, G.: Reflex Function of the Spinal Cord in Man, Brain **40**:264 (May) 1917.

13. Bastian, H. C.: Symptomatology of Total Transverse Lesions of the Spinal Cord, Med.-Chir. Tr., London **72**:151, 1890.

Muscle tone as estimated by passive movement was rather diminished than increased, and the legs did not tend to assume any special position, but would lie as they were placed. Only Case 4 was an exception to this rule. In this case there was predominence of tone in the extensor muscles so that the legs were always extended. "Automatic" bladder was established in five cases, and a small amount of urine was passed at frequent intervals. Only in Case 4 was the bladder distended, and this may not improbably be attributed to the cystitis which followed catheterization in this case. Kooy, Burr and Crothers altogether described six cases with complete physiologic transections. Five of these showed the flexion reflex, three showed the "mass reflex," and two exhibited active tendon reflexes. The four that did not show tendon reflexes were all in very poor condition. In Kooy's case active tendon reflexes were later abolished, probably as a result of bed sores.

It is striking that vasomotor and trophic changes, which develop with such rapidity in adults with severe cord lesions, were slight in my cases. Two cases showed some dryness and scaling of the skin around the thighs and buttocks where it was exposed to the irritation of the urine, but the skin was always smooth and soft where protected. Among the cases reported by other observers, only in that of Kooy did bed sores develop. No definite wasting of the muscles was made out in my cases, although the muscles of the legs felt soft as compared with the musculature of the arms.

Loss of cutaneous sensibility was apparently complete below the lesion in four cases, and there was definite diminution of sensibility in the other cases. Sensory loss was demonstrated most satisfactorily by pinching the skin at successively higher levels until the baby showed signs of pain.

CONCLUSIONS

1. Six cases of spinal cord lesions resulting from traction during breech extraction are described clinically, and one necropsy is reported. Five cases showed complete interruption of conduction in the cord.

2. The lesion was high in the thoracic segments in the surviving cases, and in the fatal case the cervical enlargement was involved.

3. Complete paralysis of the lower intercostals, spinal, abdominal and all leg muscles regularly resulted. The bladder discharged urine automatically, but there was always constipation.

4. Sensory loss was usually demonstrable up to the midthoracic segments.

5. Spinal reflexes below the level of the lesion varied in different cases, but on the average were greater than in adults. Complete "mass

reflex" was observed in two cases. Five showed the flexion reflex and active tendon reflexes.

6. In five cases the muscles of the legs were slightly flaccid on passive movement, but no atrophy could be demonstrated.

7. Trophic and vasomotor changes were remarkably inconspicuous.

8. No clinical or roentgen-ray evidence of injury to the spinal column was found. One necropsy showed no dislocation or fracture.

9. Five of the cases reported were seen within two years, and it seems probable that such cases are more frequent than is generally believed.

10. In all cases delivery was difficult, and in two cases it is certain that excessive force was employed in extraction.

I am indebted to Dr. Charles Bagley, Jr., for the notes in Case 1 and for permission to perform the necropsy, and to Dr. John Howland of the Harriet Lane Home for Children I am indebted for Cases 2, 3, 4, 5 and 6.

NEW METHOD OF RECORDING MUSCULAR TREMORS

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According to immediate causes, tremors may be divided into three groups:

1. Toxic tremors, including those due to tobacco, alcohol, alkaloids, metals, hyperthyroidism and probably those due to exhaustion.

2. Organic tremors, including tremor due to brain lesions; e. g., general paresis, multiple sclerosis, cerebral hemorrhage and thrombosis, paralysis agitans, senility.

3. Functional tremors, including tremor due to fear, hysteria, neurasthenia and other psychoneuroses (Crenshaw).

A number of graphic studies of tremors have been made.[1]

One method of graphically recording tremor movements has been by resting the finger, or object held in the fingers, on a tambor connected to a writing pen. In another method the string galvanometer is used. This of course records not the movements of the part, but the muscular contractions causing the tremor.

It occurred to me that a study of tremor movements might be of diagnostic value if a method for their graphic demonstration could be devised in which no mechanical agency interfered between the movements themselves and the graph.

Mr. Chester I. Hall, of the General Electric Company, has devised such an instrument. Figure 1 is a diagrammatic sketch made from a preliminary model, and illustrates the general principle of operating.

The primary source of light is a standard 6 volt, 21 candle power automobile headlight lamp, located in the rear compartment of the recorder case. The rays from this lamp pass through a condensing lens indicated in the sketch by L_3, and fall on the miniature hemispherical mirror A fastened to a thimble worn by the patient. The image focused by the lower lens, L_2, follows the vertical component of the motion of the source, while the upper lens, L_1, projects the horizontal component of the motion reflected from the 90 degree mirror M.

An improved recorder of the type just described, but which may be loaded and operated in daylight, is shown in Figure 2. This photograph illustrates the operation of the instrument showing the operator on the left and the patient on the right, the latter holding the tiny hemispherical mirror in the beam of light. The hood used by the

1. Eshner: J. Exper. M. **2**:301, 1897. Bornstein and Saenger: Deutsch. Ztschr. f. Nervenh. **52**:1-27, 1914.

operator shuts out external light, and enables him to look through a ruby glass in the cover and see the spots of light which are projected on the film. He is thus able to determine whether the patient is holding the tiny mirror within the field of the recorder.

The patient with the thimble on his finger should be directly in front of the source of light, but is at one side in the illustration so that the instrument may be seen.

The operation of the instrument shown is simple, being somewhat similar to that of an ordinary camera. It can therefore be operated by any physician without previous experience with or special knowledge of this type of instrument.

The daylight operated recorder is a self-contained and independent instrument. The source of light is dry cells within the cabinet. The

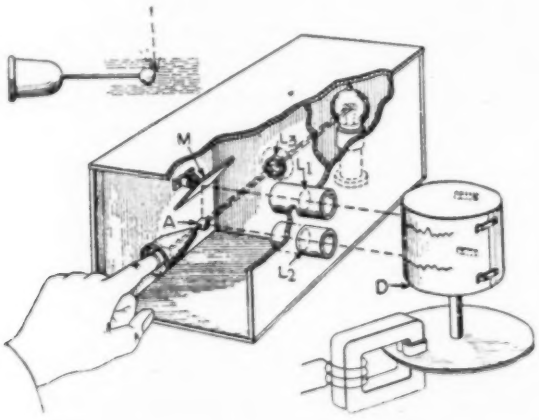

Fig. 1.—The vibration recorder with part cut away to show the paths of light beams from the course to the photographic film. The smaller upper sketch shows how the effect of a point source is obtained.

film, which is of standard size, is driven by a hand-operated crank. Constant speed of travel for the film is obtained by means of a fly-ball centrifugal governor with a clutch which releases the film driving roller when the speed exceeds a predetermined value. In operation it is therefore necessary to turn the hand-crank at any speed greater than a certain minimum and the governor and clutch insure constant motion of the film at the speed for which the governor is adjusted.

The interior of the daylight operated recorder is shown in Figure 3. This indicates the general arrangement of parts, showing dry cells, lenses, reflecting prism, etc. The automobile lamp is enclosed in the rear of the compartment shown on the right-hand side of the photograph.

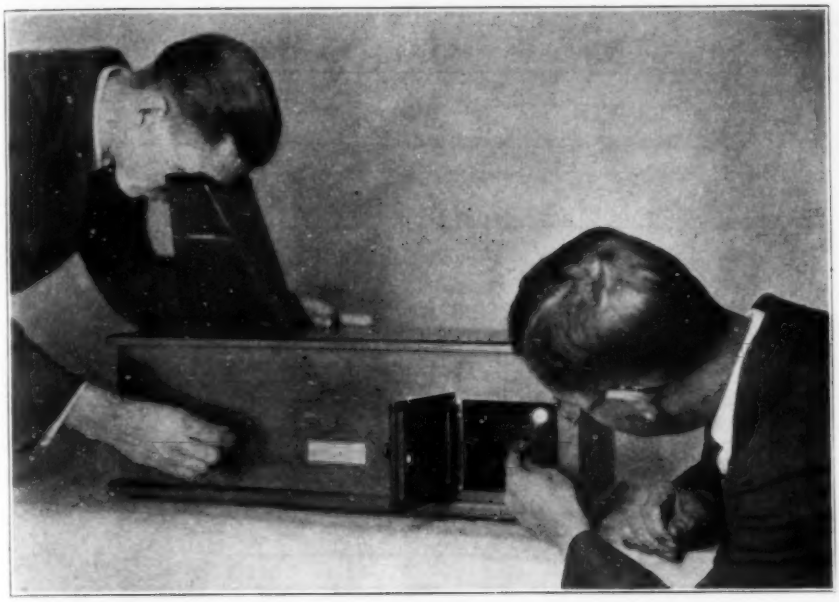

Fig. 2.—Taking tremor of patient with vibration recorder.

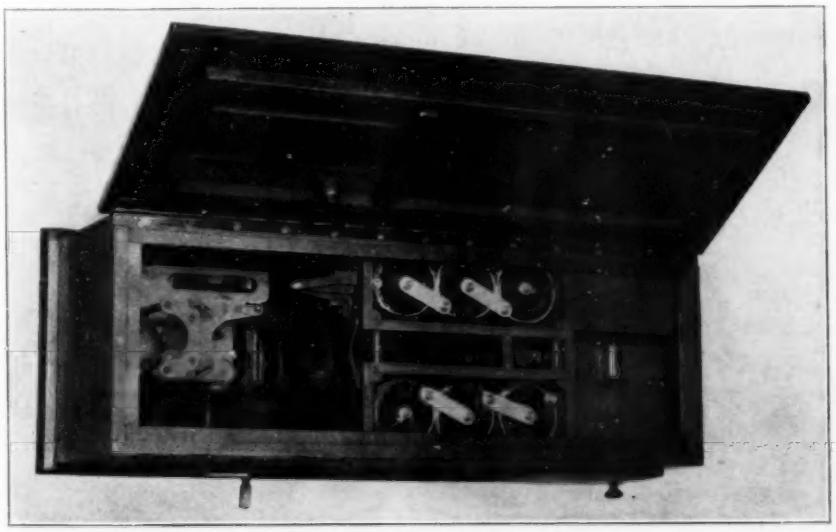

Fig. 3.—Top view of vibration recorder.

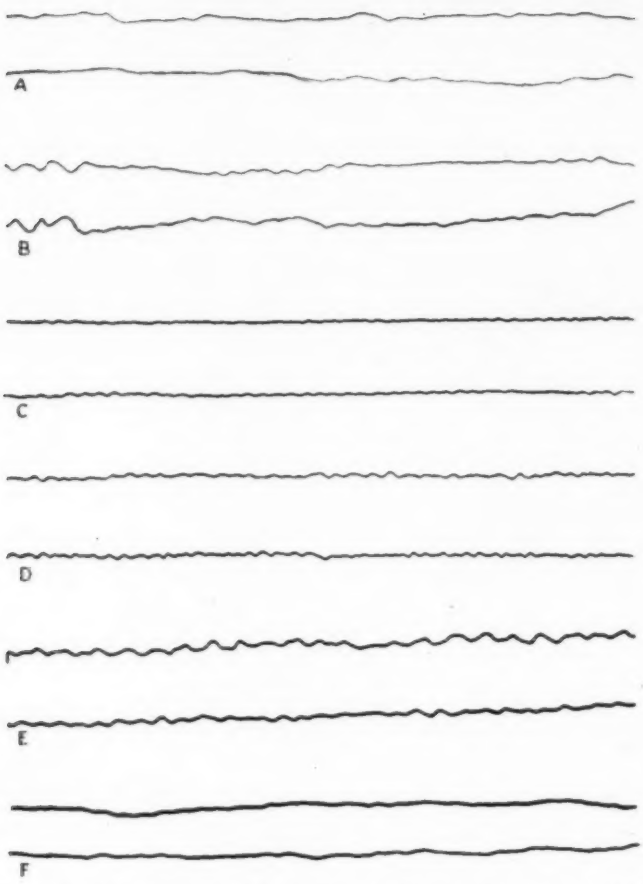

Fig. 4.—Muscular tremors in a group of normal persons. *A* is the tremograph of P. B., aged 8; *B* is that of B. B., aged 12; *C*, Dr. V. H., aged 38, when rested; *D*, Dr. V. H. when tired; *E*, Miss B, aged 75; *F*, Mrs. S., aged 102.

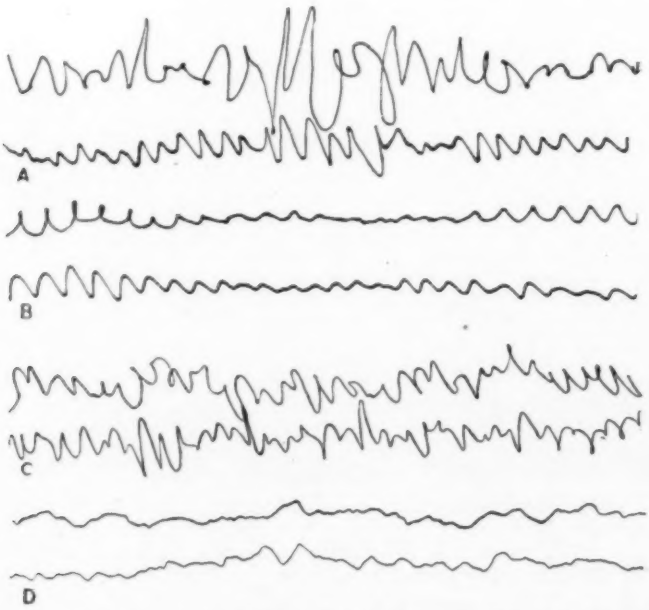

Fig. 5.—Tremor in patients with paralysis agitans. Note that the horizontal movement is much greater than the vertical. The rate of vibrations was about 8 per second. *A* is the tremograph of Dr. B., aged 32, who had paralysis agitans; *B*, Rev. D., aged 65, with the same condition; *C*, Mr. B., aged 52, with unilateral left paralysis agitans (left hand).

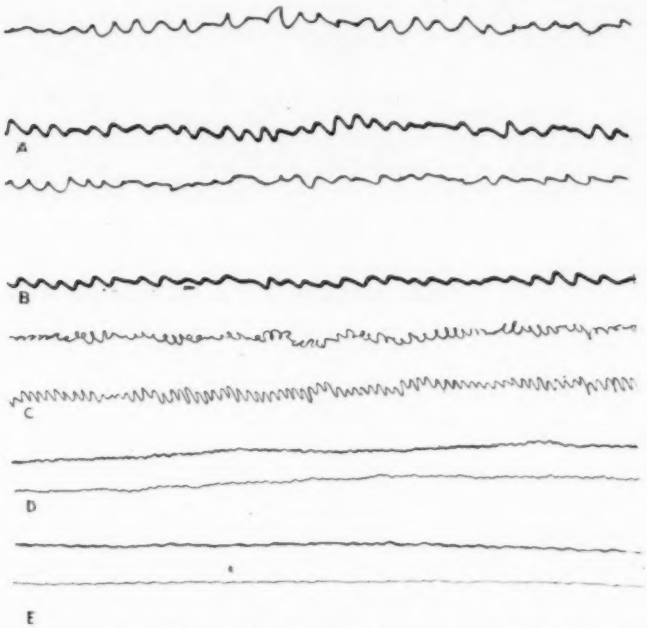

Fig. 6.—Tremor in patients with toxic goiter; rate of vibration about 11 per second. Contrast with that of patient who had myxedema. *A* is the tremograph of Mrs. K., who had adenoma (basal metabolism, +41 per cent.); *B*, Mrs. H., with exophthalmic goiter (basal metabolism, +36 per cent.); *C*, Mr. H. K., aged 33, with exophthalmic goiter (basal metabolism, +73 per cent.); *D*, Mr. H. K., six months after thyroidectomy (basal metabolism, +2 per cent.); *E*, Mr. S. D. R., aged 63, with myxedema (basal metabolism, —34 per cent.).

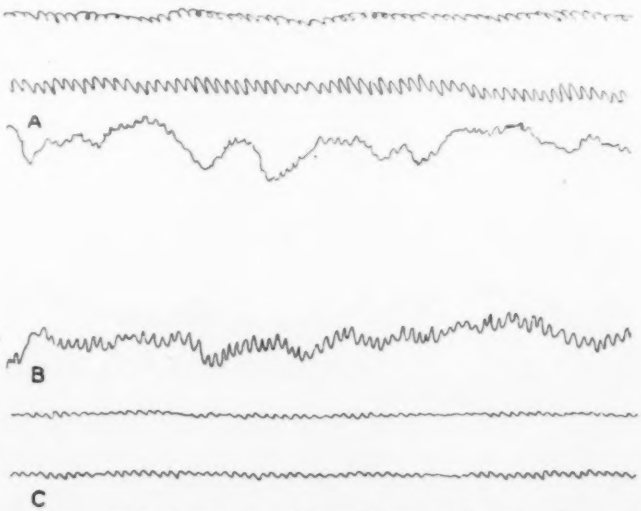

Fig. 7.—Tremograph of patients with psychoneurosis; note the resemblance to tremographs of patients with hyperthyroidism; rate about 10 per second. *A*, Mrs. B., aged 44; *B*, Mr. R. W. A., aged 29, forearm unsupported; *C*, Mr. W. L. W., aged 22.

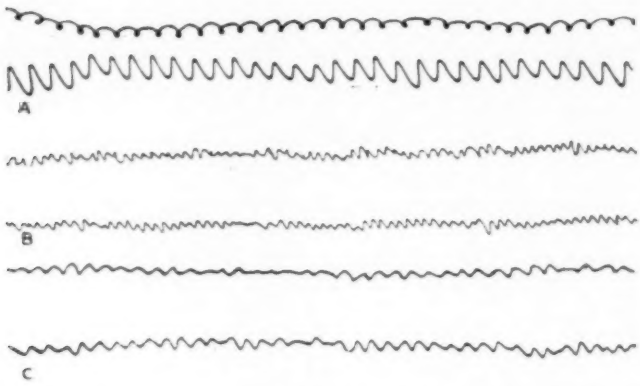

Fig. 8.—Toxic tremors. *A*, tremograph of Mrs. S. W. B., with severe diabetes; *B*, Mrs. C. E. W, with melancholia during menopause; *C*, C. W., aged 30, with chronic fatigue.

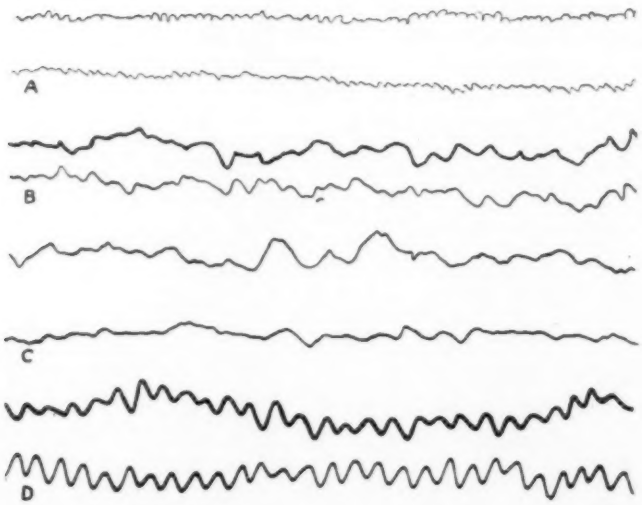

Fig. 9.—Organic tremors. *A*, Mr. F. R., with syphilis of the brain and spinal cord; *B*, M. H., aged 20, with insular sclerosis; *C*, E. C., aged 31, with insular sclerosis; *D*, Dr. R., with congenital hereditary tremor.

This lamp is controlled by contacts operated by a push button mounted in the cover. The film holder and reroll mechanism are located in the left side of the cabinet as viewed in Figure 3.

The source of light and optical system are such that the recording points of light on the film are of very small diameter and high intensity. A clear record is therefore produced, and the illumination is sufficient to make records on bromid paper.

In the graphs shown, the upper line records horizontal movements and the lower line vertical movements. Ordinates give the magnitude of vibrations multiplied by two. Chart movement is from left to right at the rate of 1 inch (2.5 cm.) per second in the negatives (with black background) and 2 inches (5 cm.) per second in the positives.

From a study of a large number of tremographs taken from carefully observed patients, it would appear that there are two great groups of tremors; first, those that are due to organic changes in the central nervous system; and, second, those that are due to exogenous and endogenous toxins. Tremors of the first group are characterized by gross irregularity in amplitude, rate and contour. Further experience may show constant characteristics of tremors which will prove of diagnostic value.

Tremors of the second group are characterized by rather rapid regular rate, regular amplitude and regular contour. Experience thus far would seem to show that toxins, regardless of their origin, produce approximately the same type of tremor, and seems to indicate that so-called psychoneurotic tremors are of toxic origin.

CLINICAL APPLICATION OF TONIC NECK REFLEXES

WITH SPECIAL REFERENCE TO TUBERCULOUS MENINGITIS *

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AND
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In a number of excellent papers Walshe [1] has called attention to the possibilities of the clinical application of the Magnus-de Kleijn tonic neck reflexes. Reference to the literature shows that early in their work Magnus [2] and de Kleijn made observations on man. Since that time a few continental workers have made similar observations in isolated instances. Finally, Simons [3] attempted to apply the physiologic facts to a great variety of cases. We have attempted to elicit these tonic neck and labyrinthine reflexes in cases of hemiplegia, tumor of the brain, and in many cases of tuberculous meningitis. We believe that the reflexes may have some localizing value and considerable diagnostic significance, particularly in tuberculous meningitis.

Early in 1909, Magnus and de Kleijn elicited certain reflexes in decerebrate animals. As is well known, the decerebrate animal of Sherrington shows extensor rigidity of all the extremities. Magnus found that rotation of the head to one side causes increased extension of the limbs on the side to which the chin is turned and diminished extensor tonus with flexion of the limbs on the side toward which the occiput points. This he called a tonic neck reflex. Since that time Magnus [4] and his co-workers have extended their observation and elicited other tonic neck reflexes as well as a number of labyrinthine reflexes. The results of these observations have recently appeared in an excellent monograph.

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* Read at the Fifty-First Annual Meeting of the American Neurological Association, Washington, May, 1925.

1. Walshe, F. M. R.: On Certain Tonic or Postural Reflexes in Hemiplegia, with Special Reference to the So-called "Associated Movements," Brain **46**:1 (May) 1923. The Decerebrate Rigidity of Sherrington in Man, Arch. Neurol. & Psychiat. **10**:1 (July) 1923.

2. Magnus, R.: Ein weiterer Fall von tonischen "Halsreflexen" beim Menschen, München. med. Wchnschr. **60**:2566, 1912.

3. Simons, A.: Kopfhaltung und Muskeltonus. Klinische Beobachtungen, Ztschr. f. d. ges. Neurol. u. Psychiat. **80**:499 (Jan.) 1923. Kopfhaltung und Muskeltonus, Neurol. Zentralbl. **39**:132 (Feb.) 1920.

4. Magnus, R.: Körperstellung, Berlin, Julius Springer, 1924.

The tonic neck reflexes are definite postural reactions which occur after a brief latent period, and which persist as long as the new position of the head is maintained. On changing this position, the postural reflexes also change. In addition to the flexion and extension of the limbs, depending on the side to which the chin is turned, the Dutch observers also found postural changes in the extremities on forward and backward and side to side bending of the head. The limbs on the side to which the head is bent (not rotated) flex, the others extend. This is the opposite of what occurs on head rotation. When the two are combined they neutralize each other, although the rotation reflexes predominate. These responses are asymmetrical in nature, i. e., the limbs of one side flex while those of the other extend. While in certain animals all the limbs flex or extend synchronously with the ventral or dorsal flexion of the head, in others the fore and hind limbs assume opposing positions. On the other hand, the labyrinthine reflexes (which depend on the stimulation of the otolith organs and not of the semicircular canals) are always symmetrical in nature; that is, when the position of the head and body is changed with reference to the horizontal plane (without altering the position of the head in relation to the body) all the limbs flex or extend, depending on whether the head is 135 degrees above the horizontal or 45 degrees below it. The latter is the position of the maximal labyrinthine influence or extensor tonus.

In addition to the simple tonic neck and labyrinthine reflexes, Magnus and his co-workers have also described so-called "Stellreflexe" (righting reflex) in contradistinction to the "Stehreflex" (standing reflex), the latter being the antigravitational posture assumed by the decerebrate animal. The animal which has been decerebrated by transection of the midbrain is capable of standing (possesses a Stehreflex) but cannot resume this position if overthrown. The animal whose transection is cephalad to the thalamus "thalamus animal" or caudad to the thalamus "midbrain animal" is capable of righting itself (possesses the Stellreflex).

It is a question whether these physiologic facts can have clinical application with reference to localization. Apparently the midbrain contains the mechanism which permits the animal to right itself. According to Rademaker,[5] it is in the red nucleus. However, the tonic neck and labyrinthine reflexes alone cannot be utilized at present for localizing purposes, because they have been found in lesions as high as the thalamic level or higher, and as low as the upper cervical cord (Simons).

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5. Rademaker, G. G. J.: The Significance of the Red Nuclei and the Other Parts of the Mesencephalon for Muscle Tonus, for Normal Attitudes and for Labyrinthine Reflexes, abst., Brain **47**:390, 1924.

METHOD OF ELICITATION

In trying to elicit the tonic neck reflexes in man, it is necessary to note the attitude of the limbs of the patient in the recumbent position. Occasionally the limbs assume postures in relation to the head which are tonic reflex responses. Ordinarily, the patient is placed on his back with the head in mid-position. The tonus of the limbs is determined by palpation. The head is then turned to one side (chin to shoulder) and held in the new position for a minute or two. After a latent period of a few seconds, the limbs on the "chin side" gradually extend and those on the "occiput side" flex. These postural changes persist as long as the position of the head is maintained. When these reflexes are apparently latent on simple turning of the head, they may be brought out in unilateral lesions by voluntary active tonic innervation of normal muscle groups, such as gripping an object ("associated movements"). It should be observed that the reflex response in the limbs may be partial in character so that only one limb or part of a limb may flex or extend. In some cases there may be only changes in tonus without obvious movement. In these instances palpations of the limbs reveal increased tonus on the "chin side" and decreased tonus on the "occiput side." In addition to flexion and extension (especially in the upper extremities), there is abduction with the former and adduction with the latter. In any case, the reflexes are more easily elicited in the upper extremities. In fact, as Walshe [6] has pointed out, the flexor element in the lower extremities may be manifested only by the presence of a Babinski dorsiflexion of the big toe.

In this connection it should be pointed out that complete decerebrate rigidity is rarely encountered in man except before death. Walshe [7] reported such a case. Ordinarily a lesion producing complete decerebrate rigidity in man is not compatible with life. The smaller diffuse or disseminated lesions usually produce partial or fragmentary pictures of decerebrate rigidity. As the occurrence of the tonic and labyrinthine reflexes depends on the presence of decerebrate rigidity, and the clinical manifestations of the latter are frequently incomplete, it follows that the Magnus-de Kleijn phenomena are likewise partial and incomplete. These reflexes have not been elicited in cases of striatal rigidity. Both Simons and Walshe have stressed this point.

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6. Walshe, F. M. R.: On Variations in the Form of Reflex Movements, Notably the Babinski Plantar Response, under Different Degrees of Spasticity and under the Influence of Magnus and de Kleijn's Tonic Neck Reflexes, Brain **46**:281, 1923.

7. Walshe, F. M. R.: A Case of Complete Decerebrate Rigidity in Man, Lancet **2**:644 (Sept.) 1923.

The Magnus and de Kleijn neck reflexes have been described in a variety of conditions. They are said never to occur in normal persons. Minkowski [8] observed them in human fetuses of two to five months removed by Cesarian section under local anesthesia. The reflexes did not occur in all the limbs, but followed consistently the movements of the head. The labyrinthine reflexes have been demonstrated during the first three months of infancy. Landau [9] described a reflex which may be interpreted as a Magnus-de Kleijn phenomenon. He observed that when an infant lying on its stomach raised its head, the back musculature and in part that of the lower limbs became rigid. This became more evident when the prone child was lifted up by the observer's hand supporting the abdomen. When the child's head dropped, the limbs became flaccid. Landau observed this reflex in infants of from 6 to 8 months, rarely after the fifteenth month, and never before the seventh week. Moro [10] described an "Umklammungs Reflex" (embrace reflex) in infants under 3 months, but apparently this does not belong to the phenomenon under consideration.

We have elicited the Magnus-de Kleijn tonic neck reflexes in a number of cases, of which we shall record a few.[11]

Case 1.—H. M., a youth, aged 18, had a tumor of the midbrain. The onset (August, 1921) was gradual, with progressive weakness of the left hand and foot. In the summer of 1923, right ocular paralysis with diplopia set in. Speech became slurred, monotonous and indistinct; urinary and fecal incontinence developed.

Examination revealed complete right and partial left external ophthalmoplegia. There was paralysis of convergence. The pupils were unequal, the left being greater than the right, and they reacted sluggishly to light. The fundi showed early papilledema. Speech was slurring and cerebellar. There were the signs, characteristic posture and spasticity of a complete left-sided hemiplegia, together with a bilateral Babinski sign and loss of all the abdominal reflexes. The spinal fluid was completely normal, as was also the roentgen-ray examination of the skull.

Magnus-de Kleijn Reflexes.—The patient was recumbent, his head in the mid-position. The left hemiplegic side showed abduction at the shoulder; the forearm was flexed at the elbow at 90 degrees; the forearm was pronated, wrist flexed, fingers extended. The lower extremity showed extension at all joints with adduction at the hip. The "associated movement" (elicited by a strong grip of the right hand) produced increased adduction and an advance of the left arm, extension at the elbow, wrist and fingers. The extensor and adductor tonus of

8. Minkowski, M.: Sur les mouvements, les réflexes et les reactions musculaires du foetus humain de 2 a 5 mois et leurs relations avec le systeme nerveux foetal, Rev. neurol. **37**:1105, 1921.

9. Landau, A.: Ueber einen tonischen Lagerflex biem älteren Säugling, Klin. Wchnschr. **2**:253 (July 2) 1923.

10. Moro: Das erste Trimenon, München. med. Wchnschr. **65**:1147 (Oct.) 1918.

11. From the Neurological Division of the Montefiore Hospital.

the lower extremities was increased, and exaggerated dorsiflexion of the left big toe was present. Turning the chin to the right produced an increased flexion at the left elbow and wrist, and flexion of the interphalangeal joints. Turning the chin to the left caused increased adduction at the shoulder and extension at the elbow. The most striking changes occurred when the "associated movement" (hand grip) was combined with the head turning. Chin to the left then elicited adduction and pronounced advance of the arm across the chest, marked extension at the elbow, pronation of the forearm and extension of the fingers. Chin to the right caused less flexion of the elbow and some abduction at the shoulder. The left lower extremity showed no definite changes. Bending of the head to the side and labyrinthine influence produced no tonus changes.

CASE 2.—S. N., aged 39, had cerebral syphilis. There was a history of syphilis and a positive blood Wassermann test. In May, 1922, he suffered from an attack of dizziness and diplopia. In June, 1923, there was mental confusion; from September, 1923, to November, 1924, a left hemiplegia gradually developed. In April, 1925, he presented the signs of a left hemiplegia (with pyramidal tract phenomena) and thalamic sensory changes.

Reflexes.—Turning of the head elicited Magnus-de Kleijn neck reflexes only when combined with gripping an object in the normal (right) hand. Chin to the left produced marked extension at the left elbow and wrist, adduction at the shoulder and advance of the left arm across the chest. Chin to the right increased the flexion of left elbow and wrist and produced marked abduction at the shoulder. Other movements of the head did not produce postural changes.

CASE 3.—A man, aged 42, developed left hemiplegia due to an embolus in the right internal capsule. He had all the classical signs of pyramidal tract involvement. The neck reflexes were not elicited with the simple head movement but promptly appeared on active prolonged grasping of an object with the right hand, the maneuver employed in eliciting "associated movements." Then turning the chin to the right resulted in adduction of the arm and flexion of the hand and forearm. Turning the chin to the left resulted in adduction of the arm and extension of the forearm and hand. The lower extremity showed no definite postural changes.

In most of these cases, the further addition of tone impulses is necessary to bring out the Magnus-de Kleijn tonic neck reflexes. This is accomplished by clenching the hand As a matter of fact, such a so-called "associated movement," as Walshe has pointed out, is in itself a postural reflex and not a movement. In many cases the simple proprioceptive impulses arising in the neck muscles on rotation are inadequate, whereas reinforcement with tonic impulses from another source serve to bring out the neck reflexes. Other tonic reinforcements, such as yawning or biting, are able similarly to activate the neck reflexes.

Magnus [12] elicited the reflex in a man, aged 69, who had an intraventricular hemorrhage. Weiland [13] obtained them in a patient who had a gumma (general paralysis), but only when he was in coma.

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12. Magnus, R., and de Kleijn, A.: Weitere Beobachtungen über Hals und Labrynthreflexe auf die Gliedermuskeln des Menchen, Pflügers Arch. f. d. ges. Physiol. **160**:429 (March) 1915.

13. Weiland, W.: Offiziel. Protokoll der Kieler med. Ges., München. med. Wchnschr. **59**:2539, 1912.

Boehme and Weiland [14] reported their occurrence in a man of 78 suffering from bilateral softening of the cortex and brain stem, and also in a woman, aged 56, who had intraventricular hemorrhage and hemorrhage into one cerebellar lobe (syphilitic endarteritis). Simons,[3] as mentioned, elicited the reflex in a number of instances not altogether well defined. He quotes Robin who elicited adduction, internal rotation and flexion of the previously extended arm on stimulation of the breast of a man soon after he was guillotined, to show that the reflex is purely spinal in nature. It is not clear how this proves the point. Simons was not able to elicit them in cerebellar or striatal disease.

THE REFLEXES IN TUBERCULOUS MENINGITIS

The tonic neck reflexes are elicited with greater frequency in children; in fact, the younger the child the more likely it is to be present. The reason for this is that the new-born child is practically a "decerebrate animal." The pallium with its corticofugal paths is not yet myelinated. Therefore it is easier for a small lesion to decerebrate an infant than an older child or adult in whom the tracts are fully developed. The observation of these reflexes in fetuses and early infancy and the occurrence of the Brudzinski sign during the first three months of life, as shown by Freudenberg,[15] furnish clinical confirmation.

Based on *a priori* reasoning, it was felt that the neck reflexes should be found oftener in tuberculous meningitis. It is well known that this disease commonly involves the base of the brain, especially the interpeduncular region, which is the midbrain area, transection of which gives decerebrate rigidity. While tuberculous meningitis also involves the convexity, it is predominantly a basilar meningo-encephalitis. Epidemic and the other suppurative meningitides are cerebrospinal, and frequently involve the spinal roots as they pierce the dura. Since section of the upper cervical roots abolishes the neck reflexes, it may be that this accounts for their rare occurrence in nontuberculous meningitis. Internal hydrocephalus also occurs with greater frequency in tuberculous meningitis, and so does the presence of the Babinski toe phenomenon. We have observed seventeen cases of tuberculous meningitis and found the tonic neck reflexes in nine. They were also present in one case of acute purulent leptomeningitis and pachymeningitis and purulent

14. Boehme and Weiland: Einige Beobachtungen über die Magnusschen Hals und Labyrinthreflexe beim Menschen, Ztschr. f. d. Ges. Neurol. und Psychiat. **44**:94, 1918.

15. Freudenberg, E.: Der Morosche Umklammungsreflex und das Brudzinskische Nackenzeichen als Reflexe des Säugligsalter, München. med. Wchnschr. **68**:1646, 1921.

encephalitis of the left hemisphere, secondary to purulent ethmoiditis. The reflexes were absent in three cases of meningococcus meningitis and one case of syphilitic meningitis.

Case 4.[16]—S. Z., a boy, aged 9½ months, had a fever and cough, followed a few days later by twitching of the face and generalized convulsions. Examination showed marked opisthotonus, rigidity of the neck, bilateral Kernig and Brudzinski signs, conjugate deviation of the eyes to the right and dilated fixed pupils. The spinal fluid contained 240 cells, 97 per cent. lymphocytes. Necropsy revealed a basilar tuberculous meningitis.

Reflexes.—The child lay on the left side, chin to the left, the right upper and lower extremities flexed, the left extended. Turning the head to the right caused extension of the right arm and flexion of the left, and vice versa; the lower extremities showed only a tendency to flexion and extension.

Case 5.—L. W., a boy, aged 3, vomited for two days, had abdominal pain, constipation, headache, generalized convulsions and sleepiness for four days. There was tuberculosis in the family. Examination showed rigid neck, bilateral Babinski, Kernig and Brudzinski signs. The spinal fluid was opalescent, had a pellicle on standing and 280 lymphocytes. Tubercle baccilli were found in the fluid. At necropsy, the base of the brain, especially around the optic tracts showed a diffuse yellow exudate and a few small grayish-white tubercles. Internal hydrocephalus was found.

Reflexes.—All limbs were somewhat rigid; chin to the right produced flexion of the left arm and extension of the right; chin to the left extension of the left arm, full extension of the left lower extremity, slight flexion of the right upper extremity and right spontaneous Babinski sign. Chin forward produced flexion of all the extremities.

Case 6.—A. B., a boy, aged 16 months, had a cough, fever, pallor and night sweats for six months. Generalized twitchings, neck rigidity and a Brudzinski sign were present. The spinal fluid contained 120 lymphocytes. The von Pirquet test was positive. Necropsy revealed: marked internal hydrocephalus; yellowish purulent exudate over lateral aspects of the hemispheres; marked pia-arachnoid thickening and yellow exudate at the base of the brain, particularly in the interpeduncular space. Diagnosis: miliary tuberculosis with tuberculous meningitis.

Reflexes.—There were slight flexion and extension of the limbs on rotation of the head, but marked increase in tonus in the limbs on the side to which the chin was turned and decrease on the occiput side, with an occasional spontaneous Babinski sign on the "occiput side."

Case 7.—A child, aged 18 months, had had a convulsion and had vomited six times two and a half weeks before admission; the convulsion had been repeated two weeks later. There were generalized twitchings, all limbs being somewhat rigid. A Brudzinski sign was present. The neck was rigid. The spinal fluid contained 210 cells, all lymphocytes. Diagnosis: tuberculous meningitis.

Reflexes.—The child lay on its back, all limbs somewhat rigid and extended, arms rotated outward, hands flexed. When the chin was turned to the right, the left arm was flexed, the left leg somewhat flexed; chin to the left, left arm extended, right arm slightly flexed.

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16. These cases were studied in the Mt. Sinai, Bellevue, Babies and Beth Moses Hospitals.

CASE 8.—A child, aged 10 months, whose illness began with convulsions, had tuberculosis of the left hilum and cavitation of the left upper lobe of the lung. The spinal fluid contained 125 cells—92 per cent. lymphocytes. The child died thirty-six hours after admission to the hospital. Diagnosis: tuberculous meningitis.

Reflexes.—The baby was lying on the right side, chin to the right, head retracted. The right upper extremity was flexed at the elbow, abducted at the shoulder, fingers extended, hand pronated. The right lower extremity was slightly flexed at the hip and knee (paradoxic reaction). The head rotated to the left, chin to the left, left upper extremity adducted and markedly extended, left lower extremity extended.

CASE 9.—A child, aged 16 months, two weeks before admission vomited and then had generalized convulsions. The child was drowsy. There was tuberculosis in the immediate family. Examination showed: rigid neck, Brudzinski and Kernig signs, fixed, dilated right pupil, right ptosis and left internal strabismus, a bilateral Babinski sign, 90 cells in the spinal fluid and 62 per cent. lymphocytes. Diagnosis: tuberculous meningitis.

Reflexes.—Occasionally the right leg was extended when the head was turned to the right. Invariably tonus was increased on the side to which the chin was turned and decreased on the occiput side.

CASE 10.—A boy, aged 3½ years, had a condition diagnosed as tuberculous meningitis. There were no definite tonic neck reflexes, but the child lay on the right side with the chin to the right. The right upper and lower limbs were extended, and the left upper and lower limbs were flexed.

CASE 11.—A child, aged 2 years, was apathetic, had a rigid neck, left central facial paresis, conjugate deviation of the eyes to the right and a bilateral Babinski sign. Diagnosis: tuberculous meningitis.

Reflexes.—Rotation of the head to the right resulted in flexion of the left upper and lower extremities; rotation of the head to the left caused flexion of the right limbs.

CASE 12.—S. Z., aged 9½ years, showed opisthotonus, rigidity of the neck, bilateral Kernig sign, conjugate deviation of the eyes, dilated and fixed pupils. Diagnosis: tuberculous meningitis.

Reflexes.—Turning the head to the right caused extension of the right upper extremity and flexion of the left, and vice versa. There was some question as to whether the lower extremities showed this sign.

CASE 13.—Diagnosis: purulent meningo-encephalitis. B. K., aged 3½ years, had pneumonia and otitis media six weeks before admission; and apathy, constipation and vomiting three days before admission. Examination revealed: Child comatose, Brudzinski sign present; left hemiparesis; conjugate deviation of the eyes; bilateral ptosis; bilateral Babinski sign; slight neck rigidity. The patient died on the day of admission. Necropsy revealed: thickened and distended dura, a large fluctuating mass over the frontal region. The subdural space on the left side was filled with a large quantity of opaque yellowish-white fibrino-purulent exudate. Diagnosis: acute purulent pachymeningitis and leptomeningitis, and purulent encephalitis of the left hemisphere; left purulent ethmoiditis.

Reflexes.—When the chin was turned to the right, right arm extended and in extreme rigidity, the left arm fully flexed; when the chin was turned to the left, the right arm was completely flexed and the left extended.

Negative Cases.—(1) A patient, aged 5 months, had tuberculous meningitis; (2) a patient, aged 3½ years, had tuberculous meningitis; (3) a patient, aged 5½

years, had tuberculous meningitis; (4) a patient, aged 5½ years, had tuberculous meningitis; (5) a patient, aged 10 years, had tuberculous meningitis; (6) a patient, aged 8½ years, had tuberculous meningitis (organisms found); (7) a patient, aged 5½ years, had tuberculous meningitis (organisms found); (8) a patient, aged 7 years, had tuberculous meningitis.

COMMENT

The frequent occurrence of the tonic neck reflexes in tuberculous meningitis, especially in very young children, leads us to believe that they may have diagnostic significance—the more so in that we have not observed them in other forms. Simons [3] is the only one who casually alludes to their presence in one case of tuberculous meningitis which he observed one day before death. Although we feel that the theoretical explanation of the occurrence of the reflexes is probably correct, it is not altogether clear why they do not occur also in nontuberculous meningitis. Opisthotonus, which is regarded as part of the picture of decerebrate rigidity, is found oftener in cerebrospinal meningitis. Bazett and Penfield [17] look on opisthotonus as a phenomenon secondary to hemorrhage or infection complicating decerebration. The Brudzinski sign, which we look on as an undoubted Magnus-de Kleijn tonic neck reflex, occurs with equal frequency in all forms of meningitis. Indeed, Magnus and Boehme and Weiland so interpret the Brudzinski sign. Brudzinski's [18] own explanation made prior to the discovery of the tonic neck reflexes was that the flexion of the lower extremities was due to the influence of the extension of the back and neck musculature on the flexion of the lower extremities. Freudenberg [15] elicited the Brudzinski sign in infants with rickets and retarded cerebral development by suddenly raising the extended arms over the head.

Binda, quoted by Tronconi,[19] first described a symptom which he believed to be almost pathognomonic of tuberculous meningitis. It consisted of a sudden raising and forward projection of the shoulder following a rapid passive rotation of the head to the opposite side. Neither Tronconi nor Binda correlated it with the Magnus-de Kleijn reflexes, but it is likely that it belongs in this category. This sign lends support to our observations. The tonic neck reflexes have been observed in a few other diseases of childhood. Magnus and de Kleijn [12] noted them

17. Bazett, H. C., and Penfield, W. G.: A Study of the Sherrington Decerebrate Animal in the Chronic as well as the Acute Condition, Brain **45**:185 (Oct.) 1922.

18. Brudzinski, J.: Un signe nouveau sur les membres inferieurs dans les meningitis chez les enfants, Arch. de méd. d. enfants **12**:745 (Oct.) 1909.

19. Tronconi: Shoulder Phenomenon in Tuberculous Meningitis, abst., Pediatra **29**:802 (Sept.) 1921; J. Nerv. & Ment. Dis. (Sept.) 1924, p. 301.

in an infant with an intraventricular hemorrhage, and in two cases of internal hydrocephalus, one in a child of 6, another in a child of 3; also in a 16 months old infant with amaurotic family idiocy. Dollinger [20] found them in one of three cases of Tay-Sachs disease. J. de Bruin [21] also found them in a 15 months old patient with this disease. This case also showed some degree of hydrocephalus. Brouwer [22] described the reflexes in one case of meningo-encephalitis. The patient was a child, aged 13 months, perfectly normal up to the age of 3 months, when its limbs gradually became rigid, the forearms pronated, and the feet dorsiflexed. There were neck rigidity, increased deep reflexes, absent abdominal reflexes and Babinski sign. The patient also showed the labyrinthine reflexes. Serial sections showed disappearance of the whole centrum semiovale, while the cortex and pyramidal tracts were less affected. The process apparently stopped at the striatum and thalamus, severing their connections with the cortex. The cerebellar cortex was also destroyed by the inflammatory process, while the cerebellar nuclei were preserved. The ventral parts of the pons and middle peduncles were almost entirely destroyed.

Finally, Stenvers [23] described a peculiar "Stellreflex" in a patient whose position of the head changed with passive rotation of the pelvis. He apparently showed no neurologic signs but was very feeble and hypotonic. He lay on the left side with the head turned to the left. When the pelvis was turned to the right, the head automatically followed to the right; when the head was forcibly prevented from following the pelvis, the eyes turned to the side to which the pelvis was rotated. The reflex was tonic and lasted as long as the new position was maintained. Magnus observed this case and believed it belonged in the category of neck reflexes.

CONCLUSIONS

The complexity of the postural reflexes as studied in decerebrate preparations makes correlation with clinical material difficult. Nevertheless, previous observers have elicited the Magnus and de Kleijn reflexes in a small variety of cases. We have repeated their observations and made a few original ones in tuberculous meningitis. Although the attempt has been merely to apply physiologic facts to bedside material,

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20. Dollinger, A.: Zur Klinik der Infantilen Form der familiären amaurotische Idiotie, Ztschr. f. Kinderh. **22**:167, 1919.

21. de Bruin, J.: Idiotie Amaurotica Familiäris Infantilis Progressiva, abst., Jabrb. f. Kinderh. **81**:460, 1915.

22. Brouwer, B.: Ueber Meningo-Encephalitis und die Magnus-de Kleynsche Reflexe, Ztschr. f. d. ges. Neurol. u. Psychiat. **36**:161, 1917.

23. Stenvers, H. W.: Un "Stellreflex" du bassin chez l'homme, Arch. Neerland de physiol. de l'homme et de l'anim **2**:669, 1918.

we feel that it is not without clinical significance. Apparently the midbrain contains a "center" for a variety of postural reflexes, and it is possible for a lesion in this region to interfere with established postures or to serve to bring out new ones. But the tonic neck reflexes alone have not sufficient localizing value. The correlation of the physiology underlying the righting reflexes with clinical material should yield important information. Certainly such observations with pathologic studies should enrich our knowledge of the physiology of the human brain.

Whether or not the tonic neck reflexes may be looked on as a sign of tuberculous meningitis, only more extended observation will determine. The observation is of anatomic physiologic and not of special pathologic significance. Any lesion of whatever nature at that particular level may yield the reflexes. However, their frequent occurrence in tuberculous meningitis is noteworthy. That they have been observed in children with hydrocephalus and amaurotic family idiocy is of interest, but not from a diagnostic point of view. Very much more extended clinical observations and pathologic correlations are necessary before definite conclusions can be drawn. But the vast number of experimental observations made by Magnus and his co-workers offer a rich and remunerative field to clinicians who would apply some of the physiologic facts to clinical neurology.

BEHAVIOR DISORDERS IN CHILDREN

PSYCHOLOGIC EXAMINATION *

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The purpose of mental examinations of children from the clinical standpoint is the determination of factors which may result in difficulties on the part of the child in adjusting himself to the environment and to find means of making possible this adjustment. In this paper I shall consider the psychologic principles involved and some of the problems encountered in the psychologic examination. It should be emphasized that the psychologic examination is an essential part, but only a part, of the complete examination. Full agreement as to what the psychologic examination includes has not been reached. By some it is erroneously considered the mechanical application of mental tests and obtaining results in terms of scores. Most writers agree, however, that much more than this is included, and that is my attitude. That the psychologist is able to contribute much information about the patient which cannot be determined by the usual psychiatric and other methods is generally recognized. Fundamentally the psychologic examination is largely the application of the principles of academic psychology in clinical observation, analogous to the use of premedical sciences in clinical diagnosis. The psychologist who has specialized in normal mental reactions approaches the child from that standpoint, while the psychiatrist, dealing with the abnormal, works from that angle. Behaviorists are concerned with a purely objective analysis of the child's reactions; those recognizing the existence of consciousness extend the inquiry to subjective mental processes, while psychanalysts observe evidence of subconscious processes.

Clinically, the psychologic examination includes the determination of the level of intelligence, native ability, or mental capacity. If there is evidence of intellectual deficiency it is necessary to determine the mental functions chiefly concerned in the defect and whether the handicap is due to temporary conditions, lack of native endowment, physical illness or faults of education. Any special ability (learning, for instance); difficulties and individual characteristics of mental processes, such as emotion, memory, attention and apperception; educational status, and within the narrow limits of present knowledge, vocational status, are

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* From the Children's Memorial Hospital, Chicago.

* Read at a joint meeting of the Institute of Medicine, the Chicago Neurological Society and the Chicago Pediatric Society, March 13, 1925.

determined and are of equal importance. This information is obtained by the use of mental tests supplemented by general observation on the character of the responses.

MENTAL TESTS

Mental tests properly used and properly interpreted furnish valuable information. Every one, whether psychologically trained or not, acquires a certain degree of expertness in the observation and interpretation of mental traits; in distinguishing the coarser differences of intelligence. Intentionally or otherwise, we set up norms or standards by means of which it is not difficult to distinguish the idiot or imbecile from the normal child. The same standards are too often used in attempts to distinguish lesser differences of intelligence. They are, however, usually based on general responses to questions, physiognomy, school grade, etc., all of which bear a relation to, but do not measure intelligence. It has often been demonstrated that these impressions are frequently, if not usually, inaccurate, whether made by parents, teachers, psychologists or physicians who observe large numbers of children. The essential difference between these observations and those made by a psychologic examination is that the psychologic observations are controlled and standardized. The environment is controlled by examining the child alone and free from distraction. The examiner secures the complete cooperation of the patient and accepts only those results which represent the latter's best efforts. Questions are asked and problems are given which have been carefully prepared and which have a definite purpose. Each problem or test has been given to large numbers of normal children under similar circumstances. The psychologist knows, therefore, how the normal child should react to each test.

Standardized mental tests have been in use for many decades. They were originally devised almost entirely by psychiatrists and educationalists as measures of special ability or ability to do some particular type of work. They were based on facultative psychology, and their use was limited. The greatest advance in psychometric methods came with the determination of intelligence in terms of mental age. In 1905 Binet, a psychologist, and Simon, a physician, conceived the idea that normal children of a given age and environment should know certain things. They felt that a normal child of a given age, in a given environment, should have the mental capacity to acquire knowledge, follow directions, and do tasks proportional to his age; furthermore, that all normal children having the same innate ability and under similar conditions should be able to do approximately the same things. Accordingly, by empirical methods, tests were devised for each year. An arrangement of tests and a standard of scoring have been found (after many revisions) which cause the median mental age of unselected children of

each group to coincide with the median chronologic age. For each year five or six tests were found which can be passed by a large percentage of the unselected children of that age, by a rapidly decreasing percentage for each lower year and a rapidly increasing percentage for each higher year. For instance; a 6 year old test is passed by 60 to 65 per cent. of 6 year old children, by 30 to 40 per cent. of 5 year old children, by 5 to 10 per cent. of 4 year old children, by 80 to 85 per cent. of 7 year old children and perhaps by 90 per cent. of the 8 year old children. The average child of 6 years, then, has a mental age of 6 years as determined by the tests. It is, of course, essential that the tests be given under proper circumstances, without subjective or objective conditions to interfere and always in exactly the same way.

Definitions of Intelligence.—The evolvement of the Binet scale and its designation as a measure of intelligence has stimulated much interest and discussion as to what constitutes intelligence. Many writers question the possibility of estimating intelligence in terms of test performance. The various definitions of intelligence usually bear little resemblance to one another except so far as they reflect the school of psychology of their author. Thorndyke [1] defines intelligence as the power of good responses from the point of view of truth or fact. Pitner [2] gives a still more behavioristic definition, in which he emphasizes the relatively new situations in life. Haggerty,[3] on the other hand, speaks of intelligence as descriptive of behavior and not of the static components of the mind. Thurston [4] speaks of intelligence in terms of the individual's ability to analyze impulses and stimuli and determine the best way of reacting to the situation. Probably Terman,[5] in stating that an individual is intelligent in proportion as he is able to carry on abstract thinking, best solves the problem, at least pragmatically. Our inability to state what constitutes intelligence does not preclude the use of intelligence tests as long as they have practical value. It has been demonstrated that the use of intelligence tests gives the most accurate and reliable index of the child's native ability or intelligence. The value of determining the intelligence rating is illustrated by the following cases:

The first is that of a 12 year old boy who burglarized a neighbor's house and took $60. At the same time he scattered notes about the

1. Thorndyke, E. L.: Intelligence and Its Measurement, Symposium, J. Educational Psychol. **12**:124, 1921.

2. Pitner, R.: Intelligence and Its Measurement, J. Educational Rev. **12**: 128, 1921.

3. Haggerty, M. E.: Intelligence and Its Measurement, J. Educational Rev. **12**:213, 1921.

4. Thurston, L. L.: The Nature of Intelligence, p. 156.

5. Terman, L.: Intelligence and Its Measurement, J. Educational Rev. **12**: 128, 1921.

house explaining in unmistakable language his hatred for the neighbor. A further illustration of his incorrect judgment in not properly attempting to escape was that the notes were in his own hand writing and mentioned specific events which led to his immediate apprehension. Because of this and other errors of judgment, it was suspected that he was of low intelligence. Mental tests, however, showed him to be in the superior intelligence group. In this case the psychologist was able definitely to state that the delinquency could not be explained on the basis of low intelligence; that further study was indicated. The second case is that of a 3 year old girl who was referred with a diagnosis of feeblemindedness. The diagnosis was made because of her general appearance and reactions. She was slow and diffident. It was difficult to secure her complete cooperation because of timidity. She appeared dull and seemed to be little interested in her surroundings. She had the characteristics which one usually associates with low intelligence. By the Stanford-Binet intelligence examination she was found to have a mental age of 4 years and 2 months, indicating high intelligence. On the other hand one often sees children who are alert and give the general impression of being of much higher mentality than they are found to be by mental tests.

The intelligence quotient or ratio of mental age (as determined by a mental age scale such as the Stanford-Binet scale) to chronologic age remains constant, at least for a number of years. The stability of the intelligence quotient is of great value in prognosis. Given a child of 8 years, with a mental age of 6½ years and an intelligence quotient therefore of 81, one can say with accuracy that five years later, with a chronological age of 13 years, the intelligence quotient will still be about 81 and the mental age, therefore, about 10½ years. One may, of course, find a difference of five points in the two intelligence quotients because of different examiners, and possibly ten points if the child has been moved from a very poor to an excellent environment during the intervening five years, but these differences are clinically inconsequential.

Other Mental Tests.—In addition to the mental age scale, there is a large number of other tests, some of which are used in practically every examination. Special age level tests have been devised for illiterate, blind and deaf children, also group tests by means of which a large number may be examined at the same time. A large number of educational tests have been formulated covering all of the elementary branches of learning. Numerous performance tests have been worked out for the purpose of determining the child's mechanical ability. Motor coordination tests are employed. Likewise, there are tests designed primarily to measure the individual mental processes, such as attention, memory, association, reasoning, judgment, apperception, emotion,

imagination, volition and will. Alertness, imagery, range of information, completion, ethical perception, suggestibility, mental control, etc., are measured in terms of test performance. Lack of time precludes adequate consideration of this important part of the routine psychologic examination. One may say in general that many of the tests furnish valuable information in specific cases; some have proved to be of little or no value.

In giving mental tests certain principles must be kept in mind. Psychologic tests have been devised largely by the trial and error method, by trying many tests for various purposes and accepting those which best measure the mental process in question. The tests must have been standardized on the same group or a similar one to which the person tested belongs. For instance, one cannot apply a test which has been standardized on school children to a child who has not had the advantage of school training. Likewise, tests standardized on children cannot be given to adults. Having given the test and obtained a score further analysis of the responses is essential. This may be illustrated by the cancellation *a* test: a test of attention. This test consists in crossing out the leter *a* each time it occurs on a page of letters. Success in the test depends on the large number of subjective and objective conditions of attention, perception of symbols and motor coordination. One cannot conclude on the basis of a low score that a patient has poor attention. It is obviously necessary to determine which of the many factors involved contribute most to the score. It also must be taken into consideration that the test is completed in a relatively short period of time. The child may be able to control his attention for a period sufficient to do the test, but have very poor attention over a longer period of time.

Estimation of the value of tests is of great importance. There is always a tendency to overestimate the value of individual tests or groups of tests. For example, one often hears one test in the Binet scale designated as a test of reasoning, another as a measure of judgment, etc. Those who were in the army remember the specific as well as the broad and general conclusions drawn from single tests in the group alpha examination. Deductions of this kind are usually not borne out by further study of the individual tested. It is also contrary to the teachings of Binet. The reason mental tests were unsuccessful previous to his contribution was due to the attempt of workers to measure lower mental processes and individual character traits with specific tests. Binet started out with the assumption that this was impossible and attempted to make his tests general and nonspecific in nature.

VALUE OF CHARACTER OF RESPONSE IN INTERPRETATION OF MENTAL TESTS

The quality or character of the responses to mental tests often furnishes information more valuable than the scores. Mental tests are always given in exactly the same way, under the same conditions, even to the tone of voice. After examining a few hundred or thousand children in this way, the psychologist is able to recognize differences and peculiarities of adequate responses. This may be illustrated by responses to the following test: "What is the thing to do when you have broken something that belongs to some one else?" Approximately 90 per cent. or more of children will say "Pay for it," or something similar. One boy gave the following response; "Well, if I should break something that belonged to someone else I would think probably that I was careless. Of course, I would find out how much it would cost to buy another one and pay for it." This response is especially significant because it was characteristic of his responses to all tests, including those of mechanical ability. The performance of mechanical tests was accompanied by a large number of unnecessary movements on completing each act. This unusual type of response indicated the involvement of a large number of apparently irrelevant and unnecessary associations in giving each response; also an increased reaction time. Obviously this type of thinking was a handicap to the boy and a factor that contributed to his failure in adjusting himself to his environment.

Another problem met in the psychologic examination of children of school age is the determination of the educational status. It is still the habit in many schools to advance the child one grade each year regardless of his ability to do school work. Assuming that other factors do not interfere, a child with the mental age of 6 to 6½ years should be able to begin first grade work; with a mental age of 10 to 10½ years, fourth grade work, etc., regardless of the chronological age. School tests have been devised and standardized which are of value in determining in a rough way the child's knowledge in school subjects. It is obvious that a child 10½ years of age in the fourth grade, who has a mental age of only 8½ years, and who therefore is unable to do more than low third grade work, will, through inability to keep up with his classes, have difficulty with teachers, lose interest and may become antagonistic to school work. This is one of the common causes of school maladjustment, and furnishes an excellent stimulus for the development of behavior difficulties. It is logical to place the child in the second or low third grade. Children having a mental age above their chronologic age —of 12½ years in the foregoing case, for example—present a different problem. It is questionable whether this child should be placed in the corresponding grade, the low seventh. Although able to do the work

from the intelligence standpoint, he would be placed with children more mature as far as personality is concerned. Many men consider it more advisable to supply supplementary work to occupy the extra time not required in doing the lower grade work. In regard to children with lower intelligence quotients, it has been fairly well demonstrated that an intelligence quotient of 90 or below precludes the successful completion of academic high school work. An intelligence quotient of 90 to 100 usually indicates difficulty in doing high school work. These facts are of great value in formulating the training program of any child, especially one showing behavior disturbances, in whom the progress should be well within the range of ability.

The psychologic examination of abnormal children brings up many additional problems. Failure to give adequate responses may be due to factors other than lack of intelligence and environmental disadvantages. Among the more common causes are difficulties of attention, association, apperception and comprehension. Disturbances of attention are most frequently found in the hyperactive child who has physical as well as mental difficulty. This is well illustrated by the case of a 7 year old girl who was under observation for three months at the Children's Memorial Hospital. She was admitted because of jerky, sporadic movements, a skip-hop gait, what was described as nervous crying and difficulties of control at home and in school. The onset had been gradual two years previously. Physically, marked hyperactivity, peculiar crowing noises at times and the gait mentioned were found. Except the restlessness, all of the symptoms were intermittent in character. The psychiatric examination was negative except that many tics were noted. By the Stanford-Binet intelligence examination she was found to have an intelligence quotient of 91, placing her in the low adequate group. Rote memory for digits was good. She was restless but cooperative. Her attention was poor but controllable. The most marked difficulty was in recalling well-known responses. Throughout the examination the reaction time was irregular and often markedly prolonged. At these periods there was evidence of great difficulty in thinking. At times she stated that she knew the response and would give it after a variable length of time. At times she appeared perplexed. The difficulty was present when responding to tests far below her mental age, and to which there was no question as to her knowing the answers. In addition to her poor attention, there was marked difficulty in giving associations which were already well formed. It is logical to assume that this was one cause of her behavior disturbance.

Much work has been done on apperceptive ability, using the Healy Picture Completion Test No. 2 as a basis for measurement. It has been found that there is marked individual variability that does not

correlate with intelligence, but which does correlate fairly well with conduct in some cases.

The value of the psychologic examination of abnormal persons is again illustrated by the following unusual and interesting case of post-encephalitic state. There was a history of personality changes following a severe attack of influenza, as well as cranial nerve changes, athetoid movements of the body and extremely slow, hesitant gait. Responses to questions were usually markedly delayed or not given. Presumably for this reason, the trouble had on two occasions been diagnosed dementia praecox and mental deficiency was suspected. By the Stanford-Binet scale he was found to have an intelligence quotient of 104, indicating adequate intelligence. It was observed that tests not performed correctly were properly done when the directions were repeated a sufficient number of times and the proper amount of time given. His attention was good. He was cooperative. The essential finding was slowness of comprehension. An attempt was made to give an association test, for example, but in three quarters of an hour it was impossible for the patient to comprehend the nature of the test, definitions being invariably given instead of associations. After comprehending the nature of a test adequate responses were given to similar tests with normal reaction times.

The effect of disease processes on intelligence has not been satisfactorily determined. Mental tests of infants are largely physiologic in character. According to the Kühlman scale, a year old child, for instance, should be able to sit and stand alone, say words of two or three syllables, imitate movements, mark with a pencil and recognize objects. In an infant with a nutritional disorder of long standing, for instance, it is impossible to state whether failure to pass the tests is due to mental arrest or to physical disease. The same question comes up in considering the etiology of mental deficiency in a child who gives a history of meningitis or encephalitis in infancy. I think one may say that there is no positive evidence that diseases not directly affecting the central nervous system of infants, such as nutritional disorders, affect intelligence. On the other hand, one often sees cases of mental deficiency in which an attack of meningitis or encephalitis in infancy was the probable cause. This is in contrast to older children in whom intelligence is not affected by these diseases. This does not include, of course, the deteriorating psychoses, like dementia praecox and epilepsy.

The relationship of intelligence to delinquency as indicated by test performance has caused much discussion. Before the introduction of mental tests, it was popularly believed that mental defectives made up a large percentage of delinquents. Surveys of penal institutions,

however, reveal distribution curves of intelligence coinciding closely with similar curves made after giving tests to persons in the community from which the inmates have come; that is, the percentage of delinquents of superior intelligence is just as high as the percentage of persons of superior intelligence in the same community. It has been concluded from these observations that intelligence, as measured by test performance, plays little or no rôle in delinquency. In studying individual cases, however, intelligence is always considered an important factor. The person of low intelligence, for instance, is usually easily open to suggestion, has poor reasoning ability and cannot understand the complicated social situation in which he lives. He, therefore, uses different methods, may be the follower in the delinquency, and is more easily apprehended. The fact that persons of high intelligence are as frequent among delinquents as in the community at large indicates that there are factors involved other than intelligence, as measured by test performance.

We might go on at length taking up the many problems met in the psychologic examination. But since types of responses of both normal and abnormal children have not been identified or classified, the individual case study is still a large part of every examination. The psychologist is also interested in emotions, instincts, will, personality, etc.

SUMMARY

1. The psychologic examination is an important, if not an essential, part of every mental examination.

2. By the use of psychologic methods, the psychologist is able to contribute information about the child which cannot be obtained in any other way.

3. The psychologist is able to contribute the following general information:

(*a*) The native ability or intelligence in terms of test performance and expressed in mental age.

(*b*) The intelligence quotient, which is an accurate index of the rate of development and of the limits of development of intelligence.

(*c*) Any special abilities or special defects.

(*d*) Within limits, the educational ability and status.

(*e*) Point out individual characteristics of response.

(*f*) Note abnormal responses which may serve as clues for further study by the sociologist, pediatrician, educationalist or neuropsychiatrist.

(*g*) Interpret the results of the psychologic examination after considering the other factors entering into the case.

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BEHAVIOR DISORDERS IN CHILDREN

MEDICAL ASPECTS *

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The subject of human behavior may seem vague and indefinite to the man of science who is accustomed to dealing with specific phenomena. This is in large part due to the fact that human behavior is an expression of the most complex organization in the world. There are so many implications in human behavior that individual aspects of it have been developed into separate and distinct sciences, such as psychology, sociology, economics, etc. Moreover, the medical man entering this field is confronted with the necessity of considering facts and points of view which are more or less foreign to medicine. This gives rise to the appearance of disharmony if not of direct opposition among those who work on the problem. Fortunately, there is no real clash between the various departments of science. There need be no attempt to supplant the older professions which have dealt with the behavior problem; on the contrary, the development of medical interest in human behavior is part of the general attack on this problem. That medicine has definite and important interests has long been recognized. Medicine has not been ready, however, to assume the responsibility, nor is the state of psychiatry at present as advanced as it must be in order to make the effective contributions required. It is under the compulsion of necessity rather than because of any feeling of real adequacy that the psychiatrist has entered the field of behavior disorders.

In order to understand the possibilities for actual accomplishment as well as the limitations in the application of psychiatry to the problem of human behavior, it is necessary to state briefly the general scientific background. Natural science—especially the biologic sciences—up to the present century has been concerned mainly with bridging the gap between the inanimate world and the living organism. This has been successfully bridged, though not completely abolished, so that now the general laws of physical science are beginning to be applicable also within the living organism. The problem of behavior represents a somewhat different frontier, namely, that between the organism as a unit and the environment. Just as the concern of biology heretofore has been mainly the extending of the laws that prevail in the universe in general so as to include the internal arrangements of the organism, so now the student

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* Read at the joint meeting of the Institute of Medicine of Chicago, the Chicago Pediatric Society, and the Chicago Neurological Society, March 19, 1925.

of behavior attempts to extend these laws from the organism back into the environment by the analysis of the specific relations between the two. A new school of psychology has been developed which seeks to subject the behavior of living organisms to the same methods of study that have been successfully applied to the behavior of matter. Behavior in this sense, then, may be defined as the reaction between the individual and the environment. It may be regarded as an equilibrium reaction, many examples of which both within and without the organism are now well known. In the evaluation, therefore, of any particular behavior manifestation, both of the reacting factors must be considered.

The human being, as the most complex of all organisms, is the outstanding example of the most complete integration known to us. Every part is in reciprocal relation with every other part. It has been shown by L. J. Henderson and others that the physical environment is also integrated. The essential difference, however, in this regard, between the human organism and the general environment is that the former is highly complex and highly concentrated, whereas the immediate physical environment is relatively simple and very diffuse. As a result of this arrangement the human organism is capable of immediate and complicated reactions to stimuli, while the environment is relatively slow in its response to disturbance of equilibrium.

THE STRUGGLE FOR SURVIVAL

The underlying characteristic of all living organisms is the survival mechanism. Superimposed on this general tendency are the three basic qualities of all living matter, irritability, metabolism and reproduction. These three qualities are completely integrated, and through the specialization and division of labor in the human organism have assumed many forms, the general effect of which is to foster survival. As part of this survival mechanism the individual organism exerts a greater or lesser effect on the environment in modifying it to suit its own needs. Man has achieved the greatest effect in modifying the environment to fit his own needs. These modifications, moreover, are much more lasting than the individual that produced them. Man has modified the environment not only with actual structures such as buildings, streets and roads, utensils, but also in certain less tangible ways such as laws and customs, and beliefs which are devised to fit the needs of the time and which often survive beyond the needs. As a result of these modifications there has been produced an interdependence between the individual organism and the immediate environment comparable to that within the organism itself. There is one important difference, however, in the degree of integration and interdependence. The environment, being diffuse, is much less immediate in its effect and much less prompt in responding to

stimuli and changing conditions, and lags behind the individual more than do the responses within the organism. In general, however, such changes as are effected are lasting in proportion to the degree to which they are conducive to the survival of the organism. Anything that is directed against survival, or that results in interference with this tendency, is therefore harmful, and may be termed pathologic. Anything that is conducive to survival may be called health. As Prof. L. F. Barker has said,[1] "An organism may be called healthy when its reactions are fitted to its needs."

Unfavorable environmental conditions which do not exceed the limits of the possibilities of life are met by successful adjustment in the case of healthy individuals. But individuals vary in strength and in their ability to meet environmental difficulties. In a state of nature or of a very primitive social integration, the Darwinian struggle for existence is often visible. Weakness and inability to meet conditions successfully usually result in extermination, so that racial purity and strength of stock are maintained automatically. In the more highly organized communities, however, owing to the increased security of the modified environment as well as to the increasing skill and knowledge of physicians, persons who may be weak in general or in some particulars are able to survive. These persons may not be able to meet successfully environmental conditions to which the generally healthy person can adjust himself with success.

The human being is born most vulnerable of all organisms, and has less physical equipment for meeting the environment than any other of the higher organisms. This is true likewise of the nervous system. Thus the human being has probably fewer instincts than any other organism, instincts in the sense of inherent neural paths which produce specific responses to environmental stimuli. Nevertheless, certain automatic responses are necessary in order for the individual to survive. He could not survive if he had to discover de novo a complete response to sudden environmental danger every time it occurred. In order to meet the usual and recurring situations, therefore, the individual becomes equipped with responses which are so automatic that they simulate instincts. Nevertheless, it has been shown that they are not instincts but have been acquired. According to Prof. Franz Boas, the first three years of life are the time when most of these automatic responses are acquired. Professor Freud has shown how important the early years of life are in the acquiring of definite behavior trends.

As part of the survival quality, the organism, especially the human organism, is much better equipped on the perceptive and apperceptive side than it is on the motor side. The individual can either fight and

1. Psychiatric Prophylaxis, Proc. Am. Psychiat. Assn., June, 1924.

struggle with the obstacle, or he can run away from it and escape. Since greater safety lies in escape than in actually clashing with the environment, the organism has become equipped with much more complicated and effective mechanisms for perceiving and avoiding than for meeting by direct action the dangers and obstacles. In the more complex psychic responses this has led to an apparent overevaluation of perception and of the sphere of reasoning and understanding. It has been easier for the individual to accept unfavorable conditions and survive than to alter them. One result of this tendency in the case of the human race is that there are always larger audiences and more criticis than there are performers.

In considering the effect of the environment and the organism on each other two factors must be kept in mind, the differences in concentration and integration between the two and the question of distance. Just as in physical chemistry it has been demonstrated that the reaction which takes place between two phases has its greatest speed and intensity at the planes of contact, so the reaction between the organism and the environment is most intense at their plane of contact, and the rate and intensity of reaction fall off rapidly in either direction away from this surface.

IMPORTANCE OF THE UNCONSCIOUS

In the study of behavior manifestations in human beings, however, it has been found that surface phenomena between the environment and the individual as a whole do not represent the entire problem. There is a further distinction of a similar sort to be made within the organism with reference to the phenomena at the line of separation between the conscious self or the ego and the rest of the organism. The greater part of the organism lies without consciousness. There is an important set of reactions which occur at this surface of contact between the conscious self and the rest of the individual. From one point of view the part of the organism which lies outside the conscious self may be regarded as environment, that is, the immediate environment of the conscious ego. The reactions on this frontier have been investigated particularly by Professor Freud and his followers. The general rules of adjustment and of differences in complexity of organization that we have described in connection with the individual as a whole and the environment are applicable also here in the differences between the conscious self and the rest of the organism. Here likewise the speed and intensity of reaction are greatest at the actual point of contact. These inner reactions, however, take place much more promptly and with much greater intensity than is characteristic of the reactions between the organism as a whole and the immediate physical environment. Thus there is the necessity for a more intimate adjustment within the organ-

ism itself, which now is expressed in terms of desires and appetites, or dislikes, pain and disgust. Within the organism deep-seated responses are acquired, which become automatic and continue to influence the adjustment between the conscious self and the rest of the organism as time goes on. It is probably because of this general relationship that the behavior reactions at the surface of the organism as a whole so often lag behind the reactions at the inner frontier. The ignoring of this factor has frequently caused perplexity in the attempt at explaining some particular act the cause for which may be located very far back in the life of the individual.

Hence in considering a behavior manifestation in a given person, on the basis of the probabilities greater emphasis must be placed on the factors within the individual than on those in the environment. Environmental factors require the greater emphasis in situations in which an entire group is affected, as for instance, in earthquakes, drought, famine and war. When a person deviates markedly in his behavior from that of the other members of his group, however, consideration must be given to the individual rather than to the environment. This leads to the conclusion that medical science, which deals with the individual, has an important interest in those behavior problems which stand out by being exceptional.

HEALTH AND DISEASE

When an individual exhibits behavior manifestations that single him out from the other members of the group, the important point to determine is whether the variation is a healthy one or whether it is due to a pathologic condition. When the variation is a healthy one the variant is in no way incapacitated in his survival mechanism. A pathologic condition, on the other hand, interferes with survival. In any given case of marked variation the likelihood of its being pathologic is far in excess of its being healthy. The pathologic factors involved in the general problem of behavior present an important point of interest for the physician.

There are two general types of pathologic conditions. The first type is represented by conditions resulting from weakness. In other words, the organism is completely formed and developed, but is weak in one direction because of disuse. The second type includes the general category of deformity, which may be acquired as the result of disease or other injury, or which may be inherited. This distinction in type of pathology is most important because of the different methods of treatment indicated in a given case.

In the former type of disability the treatment consists in raising the efficiency of the organism by proper exercise or other treatment. An example of this is the problem of malnutrition, to which so much atten-

tion has been paid of late, of anemia, or of a weak musculature. In the second category are grouped the majority of behavior problems that are presented to the medical man for consideration. Under this heading we include disorders of any of the systems of the body. The more intimately they are bound up with the functions of the nervous system, the greater is their effect on behavior. Injuries to the nervous system itself, especially to the brain, are of the most immediate importance. The effects of diseases such as syphilis, encephalitis, poliomyelitis, as well as traumatic injuries, are of primary concern. Next in importance are the disorders of the sympathetic nervous system and the glands of internal secretion. Finally, there are the disorders in systems which have less direct effect, such as the circulatory, the respiratory and the digestive systems. All these conditions are definitely organic in their nature.

There are other possibilities, however, in which the effect is not immediate or organic, but indirect and functional. Disabilities such as malformations affecting the appearance of the individual, birth marks, malformations of the ear or nose, a deformed foot or leg or hand, as well as more deep-seated disturbances, have resulted not only in influencing the effectiveness of the individual as a whole in his social life, but also have an influence on the adjusting mechanism between the conscious self and the rest of the individual. The recognition of a handicap making one peculiar or different from others, or inferior to others, may cause a great deal of difficulty in the adjustment of the ego and may lead to behavior disorders, the relation of which to this underlying cause, though it may be difficult to demonstrate, is nevertheless found to exist. Prof. Alfred Adler has developed this field of psychopathology and has demonstrated the frequent association of behavior and personality disorders with such underlying organic defects.

With every type of deformity of this sort the physician must consider the two phases of the problem: first, what can be done to alleviate the immediate organic disorder, and second, what can be done to adjust the individual as a conscious being to the presence of the disability. The cases that follow are illustrative of types of behavior disorders encountered and the treatment that has been applied.

REPORT OF CASES

A CASE OF MALNUTRITION

CASE 1.—A boy, aged 10 years and 11 months, was brought to the clinic by his mother on the advice of a general dispensary. The complaint was truancy, stealing, masturbation and lying. For two years the boy had been truant from home and school. In April of the year he was referred, he was truant from school for two weeks. He had taken sums of money, as large as a dollar, and gone away for two days at a time. He refused to stay in the house. The child's mother came to the United States from Poland eleven years

and eight months before; she was married in America. The boy, born in Chicago, was an illegitimate child by his mother's employer in Poland. The step-father, a butcher, was alcoholic and abusive to wife and children. The mother was frail, had a goiter, was fond of her children and interested in their welfare but unable to cope with the boy's delinquencies. Because of the step-father's nonsupport the mother was often forced to go to work; the home was at such times neglected. Because of the irregular income, the family diet was often insufficient, and the mother's night work and absence from the home prevented wise planning of food.

Clinical Examination.—Physically, the boy had enlarged tonsils and carious teeth, and was underweight. Psychologic examination showed that he had an adequate intelligence. Psychiatric examination showed that he was an egocentric personality.

Treatment and Outcome.—The boy's tonsils and adenoids were removed. He was put in a fresh air class in school and given milk. A recreation program was provided. This boy has not been truant since October. Good conduct is reported at home since the date that marked the commencement of this treatment.

A CASE INVOLVING HEREDITARY FACTORS

Case 2.—A girl, aged 17 years and 8 months, was referred for marked sex interest and imminent danger of sex activity. She had been adopted by well-to-do foster parents at the age of 10, from a home in which immorality and profligacy were the rule.

Clinical Examinations.—Physical examination showed absence of the hymen and distensible vagina due to masturbation. Mental tests gave an intelligence quotient of 110, superior intelligence. The girl showed considerable resistance against early recollections of childhood and marked inferiority reactions. Because she was adopted she did not recognize herself and was not recognized by the family as a real member, in spite of a superficial protestation to the contrary. She was inferior in her estimation of herself because of resistance to the new standard set up for her. She said: "I am only a slum child anyway. What's the use?" She had surrounded herself with a protective shell of hardness to prevent any spontaneous emotion.

Treatment.—The girl, as well as her family, were given a knowledge of the underlying causes of her behavior. Safe recreational outlets were provided. She was encouraged to live up to her own ideals, and by close supervision was given continuous protection against relapses, against the formation of new resistances or recurrence of old ones.

The results show progress. The patient's school work is improving; she is less aggressive, and she is willing to subject herself to guidance. This problem, a response to automatic and perhaps inherent tendencies, is probably based on deep-seated factors which cannot be corrected quickly. For a long time there must be supporting treatment by suggestion and environmental arrangement. This may be gradually reduced or even temporarily removed, but in all likelihood the girl will never be completely safe from the more ordinary menaces of social life.

A CASE OF ENDOCRINE DISTURBANCE AND MALNUTRITION

Case 3.—A boy, aged 8 years and 9 months, was referred because of stealing, lying, truancy and bed-wetting.

Clinical Examinations.—Physical examination showed the boy to be a mouth breather. The upper incisors were widely separated; both testes were unde-

scended. He had a normal basal metabolic rate, and urinalysis was negative. Sugar tolerance tests, however, showed a markedly increased sugar tolerance. Psychologic tests gave him an intelligence quotient of 94, adequate intelligence. He showed marked defect in attention.

Treatment and Results.—The physician believed that the boy was suffering from pituitary insufficiency, with a consequent arrest of development, both physically and mentally, and increased demand for sugar, this demand resulting in behavior difficulties, such as stealing money to buy candy, running away and other evidences of restlessness. It was recommended that the diet be altered to conform with the sugar needs. With an increase of carbohydrates in the diet, more food and pituitary gland supplied, running away and stealing ceased at once, although restlessness still remained. A more varied recreational program was planned, and supervision at home which would include greater variation in schedule. After two years the results are as follows: The boy remains in the group of adequate intelligence. Physical examination shows both testes in the scrotum and undernourishment much less marked. The family report that adjustment has improved, that there have been no stealing, lying, bed-wetting or vagrancy. Recently, the mother wrote: "He is very little trouble, and does what he is asked. He has not taken any money and has seemed to act like any normal boy. Except for lack of attention he is not causing any trouble in school."

A CASE OF POST-ENCEPHALITIC DISTURBANCE

CASE 4.—A girl, aged 5 years and 3 months, was brought to the clinic with complaints of stealing and extreme temper tantrums, which included head-banging, screaming, kicking and swearing. She would lie down on the street car tracks frequently and refuse to move when a car came. The parents, recent immigrants from Hungary, were economically self-maintaining. Discipline was inconsistent. A brother and sister presented no problems. At the age of 5, an acute onset of behavior difficulties appeared immediately after a serious illness diagnosed as influenza. Following this illness a complete change in personality was noted. There had been no school difficulty before this time, but the girl was later dismissed from two schools after a few days as incorrigible.

Clinical Examination.—Physically, she was anemic, and had sluggish pupillary reflexes. Her intelligence quotient was rated at 114, superior intelligence. A diagnosis of postencephalitic condition was made.

Treatment and Results.—Explanation of the situation was made to the parents. They, however, proved incapable of treating the child in the home. Foster homes were found in which social service supervision was given. After a period in which the child was in the Detention Home, the Psychopathic Hospital and the receiving home of a child-placing agency, she was returned to her own home and reentered in school. Up to this point, improvement was extremely slow. The parents were again given instruction concerning her condition, and ceased to demand the same performance required of the other children. Although not suitable in type, the subnormal room of a public school offered the only disposition which the Board of Education could make of her case at present. Her attendance is regular, and she is making steady progress in school. Although restlessness continues, temper displays have decreased, and stealing his disappeared. This treatment has been intermittent over a period of four years.

BEHAVIOR DISORDERS IN CHILDREN

PSYCHANALYTIC STUDIES *

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The various methods of approach to the study of behavior problems indicate that there is no exclusive method—all lines of investigation must be followed. This has necessitated a division of labor at the Institute for Juvenile Research and the development of special skill in the following divisions: social work, mental tests, achievement and special abilities tests, physical examination, psychiatric examination. The psychiatrist utilizes the findings in these fields, correlates them with the results of his mental examination, attempts to isolate the significant etiologic factors, i. e., explain the difficulty, and outlines a program of treatment.

I shall discuss the specific application of the method of investigation which we call the psychanalytic; not because it follows a prescribed technic of treatment or analysis, nor goes deeply into the details of the psychic life of the patient, but because attention is focused on certain sources of maladjustment chiefly emphasized by investigations of psychanalytic schools. Without the benefit of these investigations, we should be at a loss to explain the motivation of misconduct in many cases.

That as an exclusive method of investigation without correlating all the findings it would fall short of explaining the difficulty presented, is illustrated by the following case seen at the Institute in March, 1920:

CASE 1.—A boy, aged 14, expelled from school because of "obscene conduct" in the classroom of a year's duration, whose general behavior was characterized by a gentlemanly attitude toward his mother in contrast with wild sex boasting and profane language with boys at school, appeared indifferent to the psychiatric interview except to a question relating to his masturbation phantasy. He finally yielded his "guarded secret" in the second interview. During the masturbation he had visual imagery of coitus with various school girls. The image of the mother often came into this picture and was the "guarded secret" to which alone he reacted with apparently strong feeling.

Because of such findings, stress was placed in this as in other cases on the relationship between mother and child as a fertile source of mental difficulty. The father had died when the patient was 4 years old. There were no other children. The mother, who kept closely to home, treated the boy more like husband than son. She would coax him to stay in bed mornings while she fed him his breakfast. She appealed to him always as her "little man" and "protector." During the past year they had gone to dances together. Physically she was of smaller stature than he, and his reaction to her as dancing partner was as to another girl.

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* Read at a joint meeting of the Institute of Medicine of Chicago, the Chicago Neurological Society and the Chicago Pediatric Society, March 19, 1925.

That the boy's delinquent behavior was in part, probably the greatest part, an expression of this conflict, seems logical and fits into the theory of psychologic compensatory delinquency. His sex delinquencies would be considered as a spurious escape from the horror of the mother relationship, a flight because of shame, and a diversion of the sex energy into more (to him) acceptable forms. The normal psychology of shame is bound up in this reaction. Indeed a lack of emotional reaction in this connection is now considered evidence of severe mental difficulty.

Further investigations showed that: (1) the boy's father had died of syphilis of the brain; (2) the patient had unequal although reactile pupils and unequal knee reflexes; (3) his spinal fluid Wassermann test was 4 plus; (4) he had some physical secondary sex characteristics—in general a somewhat effeminate physique; (5) since the age of 5, he had had attacks of nausea and vomiting with nosebleed about three or four times yearly; (6) mental tests showed normal intelligence.

We have now a series of findings relating to: disease (congenital syphilis); inherited or acquired constitution (effeminate make-up), and inherited intelligence (presumably a superior or inferior intelligence would vary this reaction)—that are basic or at least in some correlation with the other delinquency-producing factors.

I prefer in this article to state problems rather than to tabulate therapeutic end-results in order to indicate the factors that must be isolated in behavior problems, especially the factors emphasized by the analysts, and the necessity of correlating them always with studies more generally medical and psychiatric.

As in the case cited in which the problem seemed to focus on one definite conflict, the Oedipus complex, I have selected another case in which also one factor loomed larger than all the others in explaining the difficulty, this time a case of definite organ inferiority.

CASE 2.—A boy, aged 15, had been operated on for hypospadias in early childhood. Because of a complicating infection the penis had to be amputated, leaving only a short stump about 1.5 cm. long, which flared bell-shaped at the end, the urethra ending at the penoscrotal junction. The other physical findings being negative, we assume that the all-important physical basis for untoward behavior was the deformed penis.

In immediate and fairly certain relationship with this deformity are the following data: 1. Because of the shape of his penis, he was nicknamed "Liberty Bell," a constant reminder of his deformity. 2. He always contrasted his organ with others—further consciousness of being different. 3. In actually attempted coitus, he failed because of the deformity.

Probably arising out of the factors enumerated, although not in certain relationship, are the following: 1. He frequently expressed his hatred for all girls and occasionally for all people (impotence-derivative). 2. He said that he wanted to live alone in the woods (he had camped a great deal alone and with a younger boy), and was very good at woodcraft. 3. He acted tough—smoked, swore, etc. (compensatory reactions?) 4. He played an inferior rôle

in the gang, stealing at the suggestion of the leader, yet was only a "nursery tough." In spite of his escapades, he always managed to come home at 8 o'clock.

However nicely this series of complex reactions has issued from the one assumed basic cause, we have also each of the following factors to contend with: 1. He is an adopted child whose foster mother died. 2. He lives with his foster grandmother, who has babied him. 3. He has more spending money than the others. 4. He is unattractive in appearance. 5. He is naturally dull (intelligence quotient, 80). 6. He failed in his studies. These social, physical and psychologic data are also definitely related to the difficulties mentioned, and must also be evaluated in order to understand the dynamics of this boy's behavior and the rationale of treatment.

In mental hygiene in which the problem is largely prophylactic, the method of examination is the same, and the factors although not so severe are equally complex. The first boy whose case was cited, the one with the striking Oedipus complex, led to a study of the masturbation phantasy in presumably normal high-school boys as part of a general mental examination. In general, one may conclude that the normal adolescent masturbation is in the intelligent boy accompanied by heterosexual imagery, imagery in which he is having some physical contact with a girl or girls or seeing a girl naked. Typically, a boy tries to control this imagery to the extent of controlling the dramatis personae. If a sister or a mother comes into the picture, he "crowds them out," as he sometimes expresses it. That a member of the family, as in the first case, will come to his fancy at such times produces a feeling of revolt different in kind from the first revulsion of the act itself. The obvious relief that comes to the boy when such facts are brought to light and discussed frankly is one of the satisfying features of work in this field. It is this phase, not the act of masturbation itself, nor of any sexual situation for that matter, but the psychologic conditioning of the act that motivates the individual, in which the analyst is interested. As all factors in the mental and physical life are concerned, he must always be primarily physician and psychiatrist to evaluate them.

Even complex psychologic reactions are remarkably uniform, e. g., masturbation phantasy. It is for this reason, as in general medicine, that we are quick to seize on any deviation from the standard, whether that standard is pulse frequency, infantile behavior or masturbation phantasy. When therefore a 16 year old high school boy related that when he masturbated he imaged a row of naked football players and "crowded out of the picture" his two companions, it was an indication for further study. Physical findings were such secondary sex characters as female pubic hair distribution, absence of coarse hair on limbs, trunk, or axilla; a general distaste for athletics, special ability and interest in exhibition dancing and further correlated findings, making up a complexus of findings as already described.

COMMENT

I have tried to show the importance not so much of the procedure of psychanalysis as of such research in directing us to certain sources of difficulty in our delinquent or nondelinquent patients, utilizing them in the technic of the direct interview and the necessity of correlating them always with the findings in other fields—a job for which the psychiatrist alone is fitted.

DISCUSSION

DR. ISAAC M. ABT: As a general practitioner I venture to ask, what is "normal behavior"? A school boy who is unusually bright, learns easily, has great initiative and energy, may "misbehave" much more than a dull boy with little initiative. The "bad" boy may prove to be a much more valuable citizen than the "good" boy.

Physical health and ill health also have an enormous influence on behavior and adjustment. Each child should be studied and managed as an individual problem, and the pediatrician and general practitioner should qualify themselves to be adequate guides for parents and teachers, not only in the matter of physical disease and disorder, but also in the more difficult field of behavior difficulties.

PROFESSOR L. L. THURSTONE: I agree with Dr. Beverly's paper and think it exceedingly well done. First, concerning the construction of the mental age scale: If we wish to determine whether a child is bright, the natural way is to ask some questions, watch the child in action and get him to do something. Then we could make the estimate by common sense. The only difference between that method and the Binet-Simon method is that the latter is more systematic, ruling out the personal equation, so that each examiner comes to the same conclusion. Recently we have extended the same logic in working with tests for adults, notably college students. The problem there is much more difficult, because as the child approaches the adult years his general abilities become more scattered.

I have been much interested in the relation between the psychology that we are teaching in the colleges and the psychanalytic theory, and I believe that they are not so different as they might seem to be. At present we are talking two different languages. The psychiatrist does not understand the psychologist, and I doubt that the psychologist understands the psychiatrist. The psychiatrist and psychanalyst have the advantage of dealing with matter which is more interesting and perhaps socially more vital than some of the material we psychologists deal with in the college. Our memory experiments may not seem very interesting, and yet they are effective for determining what we are looking for.

My impression from much of the psychiatric literature is that the authors have little regard for scientific method. Their material is not so arranged that another investigator can set up the experiments and come to the same conclusion. That is one difference between psychology and psychiatry. I do not see any reason why it should not be possible for the psychologist to study the extremely interesting phenomena that the psychiatrist has been specializing in, and why the psychiatrist cannot adopt a little more scientific method. We should not be too severe in criticizing the attempts to introduce rigid scientific methods into these problems, because the subject is more difficult to handle than the material some of the other sciences deal with, and yet it should have our interest and our cooperation.

PROFESSOR FRANK N. FREEMAN: What may education ask of medicine, and particularly of that branch of medicine which deals with mental processes? In the first place, there is great need for the application through education of the facts and principles derived from the type of studies which we have been hearing about this evening. There are many things that account for the inability of children to do the things which their mental tests or intellectual capacity show they should do. We know from every day observation that there are other factors. Some of these other traits may be well within the normal. Their exact nature is uncertain. There is obviously great need on the part of education for contribution to this general field. We cannot properly adjust ourselves to the children, and the children to their environment, unless we do make this type of study.

I also believe that we should enlarge the field of behavior that should be considered in this connection beyond what is conventionally known as abnormal behavior. I am not sure that we can draw a sharp line between normal and abnormal behavior.

From the standpoint of the psychologist, who is a layman in this field, it seems that there is a less sharp distinction between normal and abnormal behavior than we have assumed, and it is these borderline conditions that need our study.

This type of investigation requires the type of cooperation which is represented in the Institute for Juvenile Research. We have the psychologist on the one side making intelligence tests. He has a certain definite, clean-cut field. On the other hand, we have the pathologist studying a clear-cut type of abnormal behavior, and between these two the type of which I am speaking falls. This situation will have to be met by certain individuals familiarizing themselves with the psychiatrist's work and with the psychologist's work, so that they can be brought together.

Finally, I believe that we need also to some extent to give to the teacher in his training some comprehension of the type of difficulties that are likely to arise from maladjustments, so that the teacher can direct to special examiners children needing special examination, and can even deal with certain mild cases without referring them.

PROFESSOR THOMAS D. ELIOT: Physicians have contributed more to social work than social work has to medicine. Medicine has contributed to social work the attitude of healing without condemning. The psychiatric attitude enables the physician to deal more objectively with the patient, and therefore in a more clearsighted and effective way.

Forgiveness implies guilt, a social judgment and stigma, that block the approach to the patient. The physician, asking how he may heal, has largely replaced the priest as father confessor, and he has not only the professional advantages of privileged communications and implicit confidence, but also certain technics not available to the social worker or to the religionist.

Thorough knowledge can only be had through that degree of understanding which is beyond good and evil in the ordinary sense, being based on a deeper sort of charity or good will. But social work has given to medicine certain valuable contributions. The hospital without social work, the case history without social facts, mean imperfect diagnosis and treatment. The delinquent can no more be treated apart from his relations than the liver apart from its relations. The patient is merely the focus of interest in the case, the resultant of the combined forces that make it up.

The materialist or behaviorist can accept such a point of view without becoming in the least mystic or vitalistic. The forces playing through the body include not only those of chemical metabolism, but also the equally physical stimuli of the social environment, which often release, restrain and distort the bodily energies in unsuspected ways which a merely individualistic medicine might fail to provide for.

After all, any symptom and any disease may be viewed as behavior. The patient complains of physical discomfort due to misbehavior of his organism, and he seeks remedy because of discomfort. Another patient, whose organic maladjustment in his environment is not felt physically, also seeks remedy; but not this time from the physician. He seeks it perhaps in some sort of nervous behavior that becomes a psychoneurosis. Social misbehavior may represent merely a substitute for or attempted escape from a potential or actual psychoneurosis.

Delinquency represents efforts of the organism to adapt itself to an irksome situation, which efforts are by us considered antisocial, but which were not sufficiently inhibited by fear of the law or of group opinion to overcome the dominant impulses. Delinquency as such is not physically inherent, it is socially conditioned. Its thorough analysis, however, requires an appraisal of both internal and external forces, in order that the modifiable determinants of the body's behavior may be found and prescribed for. Thorough diagnosis may ascertain, again by logical elimination, that the amputation of a neighborhood gang or of a depraved boarder is what is requisite; or the injection of a visiting teacher or a scout troop into the case.

Just where to draw the line between mental disease and crime is notoriously a delicate matter. Here, however, I am *not* referring merely to legal battles, confused as they are by medieval and eighteenth century conceptions of psychology and morals by which the more mentally irresponsible and dangerous a man is the less is to be done to him for his own and others' protection. I am referring to the fact that it is almost an accident, so far as the behavior patterns of the patient are concerned, whether the acts involved in his attempts to adapt himself socially are judged by our community standards to be eccentric and crazy, or wicked and criminal. The psychiatric technic finds the same types underlying behavior called by either name, and proves equally useful to both, provided the patients are seen when young enough.

Already the functions suggested by Professor Freeman are being performed in many places by the psychiatric social workers and visiting teachers. Already the attendance officers, the medical inspector and school nurses, the psychologists and special class teachers, the vocational counsellors, when they are socially minded, are reaching out beyond their strict duties in order to adjust those children whose difficulties are chiefly misbehavior. The three-fold team is present in many school systems, but has not been integrated. What is needed is a recognition, first, of educational responsibility for delinquency, and second, of the administrative unity of all the special staff services which deal with the variate and unadjusted child. A bureau of adjustment and guidance, manned with physicians, psychologists, nurses and school social workers (i. e., visiting teachers) would combine the medical approach, the psychologic testing, the technic of social case work, and the project method in which the acquisition of new habits and status would be the objective. This team work made necessary by the very unity of the child himself would at the same time integrate and individualize the treatment and cure of behavior problems in their incipiency.

JULIUS GRINKER: I think there is no conflict between psychiatrist and psychologist. The latter works with something like a test tube or a stop watch in hand. The psychiatrist does not often use a stop watch or other recently devised scientific implements. He is required to make the diagnosis quickly and must get his information rapidly. It is to be hoped that in the future these two classes of workers will work together.

DR. BERT I. BEVERLY: Mental tests have been given so much consideration that their relative importance may have been overestimated. I should like to emphasize by repetition, therefore, the point brought out in the paper, that the interpretation of the reactions to the tests is often far more important than the scores. The tests furnish a method of securing controlled observations.

DR. DAVID M. LEVY: Dr. Abt has asked for a definition of normal behavior. Our concept of normal behavior is based on the dictum, "normal is as normal does." It is not based on our idea of what it should be, or on any philosophic, religious, or other "expert" notions, but on an actual study of such behavior. In principle, the determination of such standards is like the determination of any other standard, in the same way, for example, as the determination of the "normal" pulse. We have an idea based on observation and correlation of data as to normal pulse variations and are quick to focus our attention on any deviation from that norm. Until we have established such standards our work is faulty and misleading.

Our attitude toward masturbation is a good example. In the eighties it was thought from a study of abnormal persons only that such conduct was invariably associated with insanity and would itself therefore lead to insanity—the probable source of the parental threat to the son who masturbates. Only in recent years has an adequate study been made of such behavior in a normal group. This has shown that the vast majority of boys masturbate, 93 per cent. according to Peck and Well's analysis of the replies of 250 Harvard graduate students. Our recent studies of a high school group made in a neighboring town through individual examination confirms this finding. There also remained a small group whose auto-erotic behavior could not be included under the narrower term of masturbation.

On the basis of these data our concept of the normal in respect to this behavior changes entirely, for we must now conclude that masturbation is in the young adolescent evidence of normal behavior; further, that the boy who does not masturbate deviates from the ascertained norm much further than the boy who does, i. e., he is in this respect less normal.

Until we study "unselected" groups we are unable to determine how frequent or how great are the assumed abnormalities unless they are extreme. Occasionally superior children as determined by school achievement or mental tests are brought to a psychiatrist because of behavior difficulties. The psychiatrist is thereupon tempted to correlate superior intelligence with abnormal behavior. Naturally the factor of selection in his practice or in the child guidance clinic is the abnormal behavior, not the superior intelligence. A study by Terman and others of large groups of children in which the selective factor was the superiority and the criterion for normality certain social factors, like number of friends, play interests, etc., shows conclusively that superior children, typically come from superior parents, have superior opportunities and accomplishments and are more "normal" in respect to the criteria used than the so-called average children.

A STUDY OF THE VIBRATORY SENSATION *

RICHARD S. AHRENS, B.S., M.D.
MINNEAPOLIS

Recently, an effort has been made to standardize a satisfactory method for the quantitative estimation of the vibratory sensation. The first work of this type was carried out by Dr. J. L. M. Syms [1] of Guy's Hospital, London. Since then Drs. E. J. Wood,[2] H. D. Piercy [3] and others have made observations, using Syms' method. This work can therefore have no claim to originality. Its purpose is to establish further a normal, to compare my findings with those of other observers, and to establish the value, if any, of this test as a part of any routine examination.

A brief review of the subject shows that Rumpf [4] was perhaps the first to make a study of vibratory sensation and reported it in 1889. He considered the sensation confined to the skin. About six years later (1895) Trietel,[5] using a fork of 128 vibrations, and attempting to time the duration of the sensation, reported that in certain disease conditions the vibratory sensation might be impaired while the tactile sensation remained normal, thus distinguishing vibratory sensation from sensations of pressure and touch.

Since then various workers have recorded their observations and have advanced various opinions as to the source of the sensation. Egger [6] maintained that the vibratory sensation was in no way dependent on tactile sensation. Goldschneider [7] contradicted this, stating that the soft parts were also capable of perceiving the sensation, and he refused to consider this sensation as entirely separate from the sensations of touch and pressure. Bing, by anesthetizing various areas of the skin with cocain, demonstrated that the vibratory sensation was not abolished or even markedly diminished, although other forms of sensation were destroyed. Head [8] cites a number of cases in which both the ulnar

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1. Syms, J. L. M.: Brit. M. J. **1**:539, 1912; Guy's Hosp. Rep. **66**:120, 1912; Quart. J. Med. **2**:33-58, 1917.
2. Wood, E. J.: Guy's Hosp. Rep., January, 1921; Am. J. M. Sc. **163**:19-30 (Jan.) 1922.
3. Piercy, H. D.: Ohio State M. J. **19**:572-577 (Aug.) 1923.
4. Rumpf: Neurol. Centralbl. **8**:185, 1889.
5. Trietel: Arch. f. Psychiat., 1897, 29.
6. Egger: J. de physiol. et de path. gen. **3**:1, 1899; Compt. rend. Soc. de neurol., Feb. 4, 1904; Rev. neurol. **16**:345-351, 1906.
7. Goldschneider: Berl. klin. Wchnschr. **13**:353 (April 4) 1904.
8. Head: Studies in Neurology, Oxford Med. Pub. **1**:143, 1920.

and median nerves were severed at the wrist without injury to the tendons. As a result there was a loss of superficial sensation but an undisturbed deep sensibility, including the vibratory sensation. On the other hand, Barker [9] considers vibratory sensation to be the result of stimulation of the touch points of the skin. Williamson [10] maintains that the sensation is present in the soft parts and considers the presence or absence of the sensation on the abdominal wall of value in localizing cord tumors. Head [11] agrees with this.

Present knowledge concedes that the sensation may be felt in the soft parts, but that it is best recognized when contact is made with bone; it should be classified with joint sense, pressure sense, deep muscle sense, etc. To further establish its identity, Reidel and Seifer [12] gave it the name palesthesia, the absence of the sensation being termed palanesthesia.

The course of the fibers has been well established, chiefly by clinical findings. According to Head,[13] the impulse travels from the periphery to the cord through afferent fibers arising in the muscle tendons, periosteum, bones and perhaps the arteries, and entering the cord through the posterior roots. In the cord these fibers ascend uncrossed in the posterior columns without forming a secondary tract. They then cross in the fillet to the opposite side and enter the optic thalamus. That they ascend uncrossed in the cord can be demonstrated in a Brown-Séquard syndrome in which the vibratory sensation (as well as other forms of deep sensibility) is absent on the side of the lesion and present on the other side, whereas superficial sensation is abolished on the opposite side and present on the same side as the lesion. That the fibers ascend in the posterior columns is shown by the disturbance and ultimate loss of vibratory sense in such diseases as tabes and pernicious anemia. From the optic thalamus the fibers ascend to the cortex. Head [13] states that *no lesion above the point where the fillet enters the optic thalamus* will produce complete insensibility to the tuning fork, but that lesions of the thalamus and the cortex may alter the sensation. Cases of concussion are excepted.

Throughout the study of vibratory sensation various types of instruments have been used, but for the most part the simple tuning fork (C. 128) has seemed to meet with most favor. The use of such an instrument meant, however, that only the presence or absence of the

9. Barker, L. F.: Monographic Medicine **4**:136.
10. Williamson, R. T.: Brit. M. J., July 20, 1907.
11. Head: Studies in Neurology **1**:45.
12. Reidel and Seifer: Arch. f. Psychiat. **39**:50, 1903.
13. Head: Studies in Neurology **2**:591.

sensation could be determined. The knowledge that the sensation was completely abolished in certain disease conditions led to the demand for an instrument with which quantitative readings could be made. Little progress was made toward perfecting and standardizing such an instrument until 1911, when Dr. Syms [1] of Guy's Hospital, London, reported his findings in the *British Medical Journal*.

Dr. Syms used a fork devised by Edelmann of Munich, made of steel and hand forged, with 108.75 double vibrations a second. At the ends of the prongs were two overlapping metal plates, one of which contained two notches, one above the other. When the fork was at rest these apertures were not visible, but when the fork was vibrating at a sufficient amplitude and held between the operator and the light the two openings were visible, the upper one disappearing first, then the lower one, as the amplitude of the swing gradually decreased. In using the fork it was made to vibrate by striking it, then held to the light and the openings observed. When the vibration had decreased until the lower aperture had just disappeared, a stopwatch was started. The fork was then applied to a bony prominence and the patient instructed to observe closely the sensation and to indicate when he no longer felt it. At that point the watch was stopped, and the time during which the patient had felt the sensation was recorded. Thus a reading in seconds was obtained always starting from a fixed point and ending when the patient indicated that he could no longer feel the sensation.

Dr. Syms, using thirty normal cases, established with this fork what he considered to be a normal curve. Dr. Wood,[2] who had worked with Dr. Syms in London, found that in 100 cases which showed no neurologic pathology, the curve varied considerably from that obtained by Dr. Syms. This difference in the curves was found to be due to a variation in the two tuning forks used, and when this was taken into consideration, the two curves corresponded closely. In order that the work might be carried on by others, Dr. Wood had the fork which he used duplicated by Down Brothers of London. These duplicate forks still varied somewhat from the original, but the difference was much less than between the original forks used by Wood and Syms. The new forks are standardized by checking them against the original and the variation noted. This information is supplied to the purchaser of the fork.

GENERAL FACTORS INVOLVED AND AUTHOR'S TECHNIC

Before considering in detail the results which I have obtained, I wish first to mention some of the general factors involved, the technic which I have followed and the basis on which the following findings were established.

As in any test which depends on the subjective sensations of the patient, a considerable degree of error is to be expected. Further, in

this test we are not dealing with a clear-cut cessation of sensation, but rather with a gradually diminishing intensity of sensation which fades into absence. To state when the stage of very slight sensation merges into the stage of absence is at times difficult. Therefore, the value of the test depends on the intelligence of the patient and on the ability and willingness to concentrate.

Other factors which must be considered are fatigue, extraneous sounds or vibrations, as of heavy trucks on the street, and distracting factors in the immediate neighborhood of the patient.

In making the test I have tried to have the patient relaxed, either in bed or in a reclining position. Only intelligent patients willing to cooperate in the test were used for normals. Three or more readings were made over each of the following bony prominences: right and left internal and external malleoli, tibiae, anterior sacral spines, each radius and ulna, the sternum and sacrum. By taking more than one reading it was found that variations in successive readings over the same prominence were common. For the most part these variations were slight, usually not exceeding three seconds, and after considerable experience with the fork I have come to the conclusion that any difference of five seconds or more in successive readings over any bony prominence is due to lack of attention on the part of the patient or to error on the part of the examiner in starting or stopping his watch. By taking more than one reading it was possible to determine marked variations and secure the correct time by urging closer attention on the part of the patient, and it has also been possible to average the slight differences and thus probably establish a more normal reading. All of the charts are based on such averages.

In studying the normal curve I have examined to date 100 cases which showed no neurologic pathology. Some of these patients had no illness, some cases were medical, some surgical, but in every one a complete neurologic examination was made. The intelligence and cooperation of the patient were also assured.

RESULTS OBTAINED

The results are best presented in the form of a chart. Chart 1 represents the normal curves obtained by Syms, Wood, and myself. The marked difference between Syms' and Wood's curves has already been explained on the basis of difference in tuning forks. My findings are comparable only with those of Wood, since they were made with a duplicate of his work and an equal number of cases studied. The largest variations occur in the upper limits of normal, however, and this fact is of considerable importance since the chief value of this test lies in the determination of a diminished sensation.

Further analysis shows that while this curve varies considerably from that obtained by Wood, still it is only the occasional case which makes it do so. This is demonstrated in Chart 2. From this it is seen that between 72 and 96 per cent. of all the readings over each point were within the limits for normal as established by Wood, and that it was usually a single reading in one case or another which caused the marked disparity between our curves.

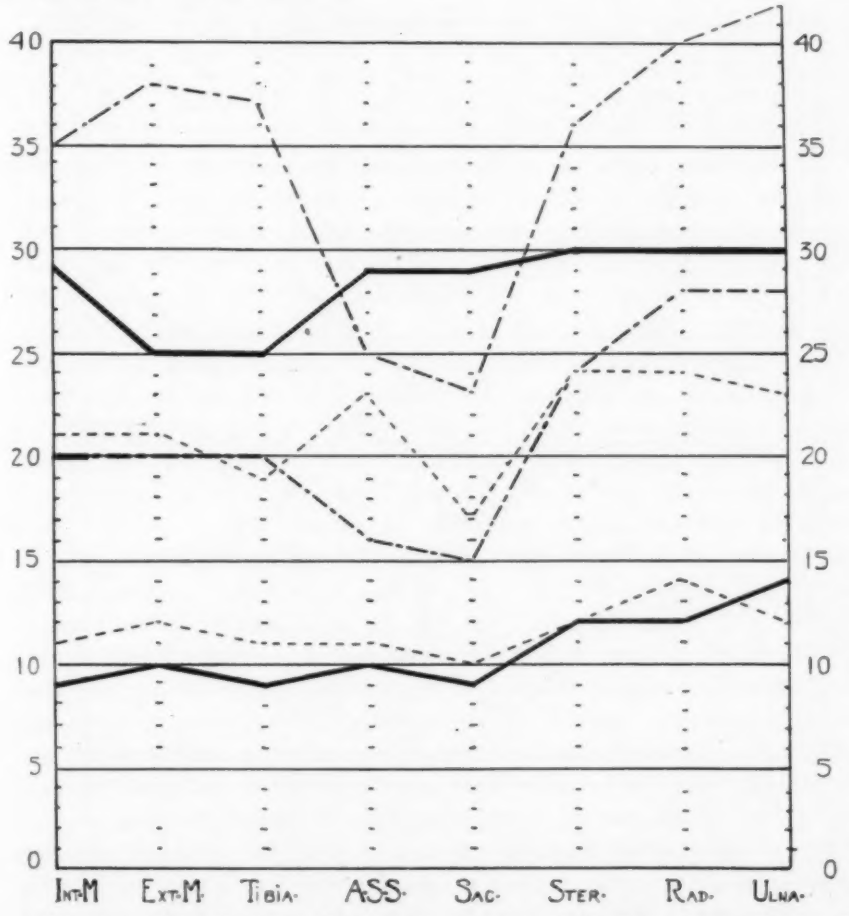

Chart 1.—Normal curves of vibratory sensation obtained by Syms, Wood and Ahrens. The long and short dash lines represent the upper and lower limits as established by Syms in thirty cases; the short dash lines those established by Wood in 100 cases; the solid lines the author's findings in 100 cases.

Further study of these normals shows that:

1. The majority of the cases showed an increased sensation in the upper extremities as compared with the lower.

2. Only fifteen cases showed the sternum as more sensitive than the ulna and radius.

3. As in Syms' and Wood's series, the majority (68 per cent.) showed a decreased sensitivity over the sacrum.

4. Almost invariably the sensation was felt longer on one side of the body than on the other. Usually, however, the difference was not more than a few seconds. In this respect the following details were noted:

	Lower Extremity Cases	Anterior Sacral Spines Cases	Upper Extremity Cases
1. Right side greater than left	28	40	49
2. Left side greater than right	22	36	27
3. Crossed (including identical)	50	24	24

From these figures it would seem that there was no predominating increased sensitivity in either right or left lower extremity or anterior sacral spine. In fact there were more identical readings over the anterior sacral spines than over any other point, which may be accounted for by the rather close union of these two bones. In the upper extremity, however, the right side showed a definite increased sensitivity. Since no record was kept as to whether the patients were right-handed or left-handed, no definite conclusions can be drawn, but since most people are right-handed, and since deep sensibility is an important factor in coordinated movements, the figures would suggest that deep sensibility was more acute in the extremity most used. This point should be kept in mind during future work.

In the field of neurologic pathology perhaps the most striking findings are found in tabes dorsalis. It had been known for some time that in this disease the deep sensibilities were progressively impaired. Williamson,[10] whose work was entirely qualitative, stated that in tabes the vibratory sensation was sometimes lost in the legs before the other forms of sensation were disturbed. Syms [1] pointed out that Mott and others had shown that the vibratory sensation might be absent over the sacrum and present over the other bony prominences in certain disease conditions of the central nervous system. It remained, however, for Syms to demonstrate quantitatively that in tabes the vibratory sensation was the first form of sensation to be affected. Dr. Wood,[2] in a study of eighty cases, stated that there had been no exception to the rule "that in the lower half of the body there always occurred a reduction in the time of appreciation of vibrations of fixed amplitude" and that "this reduction in time was singularly constant over the sacrum." He further pointed out that in early tabes the first and sometimes the only finding was a diminished vibratory sensation over the sacrum which gradually became lost as the disease progressed. It was then impaired over the lower extremities, and finally entirely lost up to and including the sacrum. Piercy,[3] who studied fifty cases using a duplicate of Wood's

fork, reported similar findings. Of his fifty cases only three gave a reading of ten seconds (low normal) over the sacrum. In these three exceptional cases the patients had pulmonary tuberculosis, which may have been a coincidence. Of proved cases of tabes I have examined to date twenty-three. In none of these cases have I found a normal vibratory curve. My findings have agreed entirely with those of Syms, Wood and Piercy. The upper extremities in my cases have

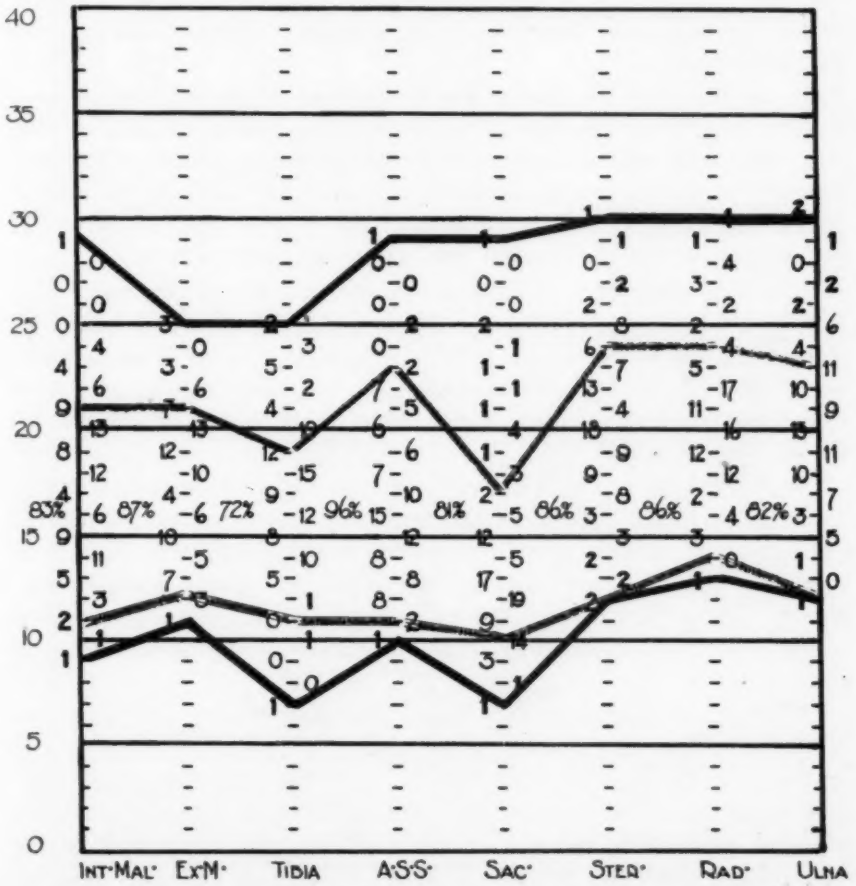

Chart 2.—Percentage of comparison of Wood's and Ahrens' series of 100 cases. The solid lines represents the upper and lower limits in the author's series; the shaded lines represent the upper and lower limits in Wood's cases. Opposite each subdivision representing one second are placed the number of cases which gave that respective reading.

rarely shown any impairment and often the sensation has been felt longer than the normal established by Wood.

Chart 3 shows a composite curve of the cases examined. This brings out very well the sacral dip in the early cases and the varying

degrees of impairment in the lower extremities, ranging from normal to total absence, also the constant normal curve in the upper extremities.

Several cases of interest may be cited as examples. One patient who gave a history of infection, and whose blood and spinal fluid were positive, the latter with a cell count and colloidal gold curve of the tabetic type, showed no neurologic findings other than an absent vibratory sensation over the lower extremities, anterior sacral spines and sacrum. Seeing this patient for the first time, the diagnosis of tabes would not have been considered if the vibratory sensation had not been carefully studied.

In another case the history was that of pain in the left leg following the course of the sciatic nerve. Neurologic examination showed slightly irregular pupils with a moderately sluggish reaction to light. The deep reflexes in the upper extremities were normal. The right patellar reflex was sluggish though definitely more active than the left. The left thigh was 5.5 cm. smaller than the right, and the left calf 5 cm. smaller than the right. Sensation was normal throughout except for impaired vibration sensation over the sacrum. There was a history of alcoholism, but no history of specific infection. Spinal fluid examination, urged on the basis of the vibratory curve, showed 74⅓ cells, positive Nonne and Pandy tests, a four plus Wassermann reaction to all antigens and a colloidal gold curve of 345554220.

In several other cases a normal vibratory curve was reported, but on examination by the quantitative method there was found to be a marked impairment or absence over the anterior sacral spines and sacrum, or possibility only over the sacrum. The report of normal findings were true as far as they went. The point is that when the sensation was studied over the sacrum, a point rarely examined, the impairment was discovered.

These cases emphasize the value of this test in diagnosis. No test is infallible, however, and this is especially true when one must depend on the patient's subjective sensations. Thus, one case showed a vibratory curve characteristic of tabes. The history and neurologic findings were not particularly suggestive. Several days later a repetition of the test showed a normal curve. Another patient admitted exposure but denied infection. His entire history, from pain in the heel to inability to concentrate, suggested syphilis of the central nervous system. His neurologic findings helped to confirm the suspicion, and his vibratory curve was characteristic of tabes. The patient was sent to the hospital for spinal puncture and there, before puncture was performed, a reexamination showed the vibratory curve to be normal. Two blood Wassermann tests were normal, the spinal fluid was normal throughout, his vibratory curve was again normal at a later date, and all of his symptoms and most of his neurologic findings cleared up under rest and treatment with phenobarbital.

These cases emphasize the fact that while a vibratory reading showing a sacral dip is suggestive, it is by no means pathognomonic, and only when it is present on repeated tests can it be regarded as final. Total absence over the entire lower extremities with a normal reading at a later date has not been encountered.

Another disease in which the quantitative estimation of the vibratory sensation promises to be of value is in pernicious anemia. Here again

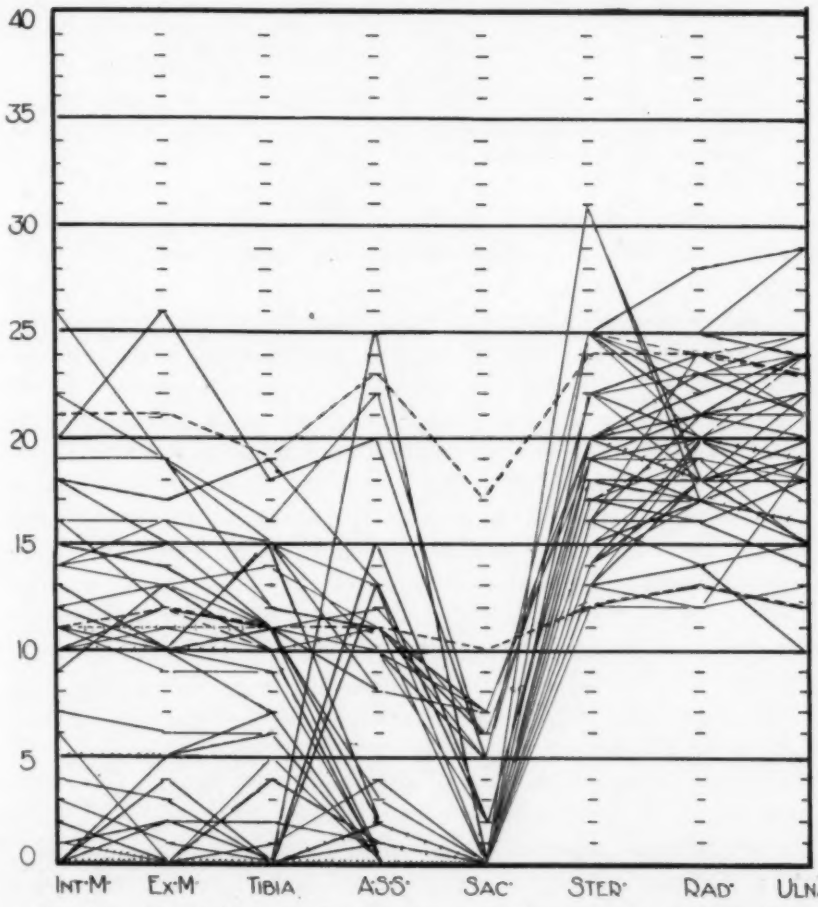

Chart 3.—Composite curve of vibratory sensation in cases of tabes dorsalis examined.

it has been recognized for some time that the vibratory sensation as well as the other forms of deep sensibility were early disturbed. In 1913-1914 A. Déjerine, J. Déjerine, Jumentie, and Mouzon [14] first

14. Déjerine, A., and Mouzon, J.: Rev. neurol. **22**:382 and 388, 1914. Déjerine, J.: Compt. rend. Soc. de biol. **75**:554-556, 1913. Déjerine, J., and Jumentie, J.: Rev. neurol. **271**:273, 1914.

clearly established this fact. Since then other observers have called attention to this neurologic finding. Thus Cadwalder [15] reported eight cases, in all of which, except one, the vibratory sensation and the sense of position were diminished or lacking. Woltman [16] reported 150 cases of pernicious anemia in which the joint or vibration sense was impaired or absent in 92 per cent. of the cases showing neurologic symptoms.

Drs. Hamilton and Nixon [17] reported their findings in forty-one cases. In these the vibration sense was found to be as follows:

Diminished in upper extremities	9
Diminished in lower extremities	1
Diminished in both extremities	2
Absent in upper extremities	0
Absent in lower extremities	20
Absent in both extremities	3
Normal in both	5

No mention was made of the type of fork used or of their technic, but it is evident that an attempt was made to estimate quantitatively the degree of disturbance.

In the quantitative study, Dr. Syms, who did not report the number of cases observed, and Dr. Wood, who reported five cases, and Dr. Piercy, who reported one case, all note the general tendency of the curve to be from an absence or an impairment of the sensation in the distal portions of the lower extremities to a gradual approach to normal in the upper extremities. That this type of curve existed in the majority of the cases studied by the qualitative method and reported by Drs. Hamilton and Nixon [17] is brought out by their statement that in the twenty cases which showed an absence of the sensation in the lower extremities "this absence extended in most of the cases as far up as the malleoli and many times over the tibia and patella."

In my series of seventeen unquestionable cases, the findings have been quite constant. Chart 4 demonstrates very well the type of curve which has been described. In comparing my findings with those of Drs. Hamilton and Nixon, I would call attention to the following: (1) No patient whom I have examined has shown an impairment in the upper extremities alone; (2) no patient in my series showed a loss of vibratory sensation in both hands and feet; (3) no patient so far examined has shown a normal vibratory sensation. One patient in my series showed a diminished sensation over the

15. Cadwalder, W. B.: Diagnosis of Subacute Combined Sclerosis of the Spinal Cord Associated with Severe Anemia, Abstr., J. A. M. A. **66**:2035-2036 (June 17) 1916.

16. Woltman, H. W.: Am. J. M. Sc., March, 1919, pp. 157 and 400.

17. Hamilton, A. S., and Nixon, C. E.: Sensory Changes in Subacute Combined Degeneration of Pernicious Anemia, Arch. Neurol. & Psychiat. **6**:1 (July) 1921.

ulna and radius, but the sensation was proportionately lower in the lower extremities so that the characteristic rising curve obtained. Four cases in my series showed what we have already considered to be a characteristic tabetic curve. Of these patients, three were examined while having a marked remission. The type of curve obtained might have been influenced by this fact, but unfortunately no opportunity to examine these particular patients during an exacerbation

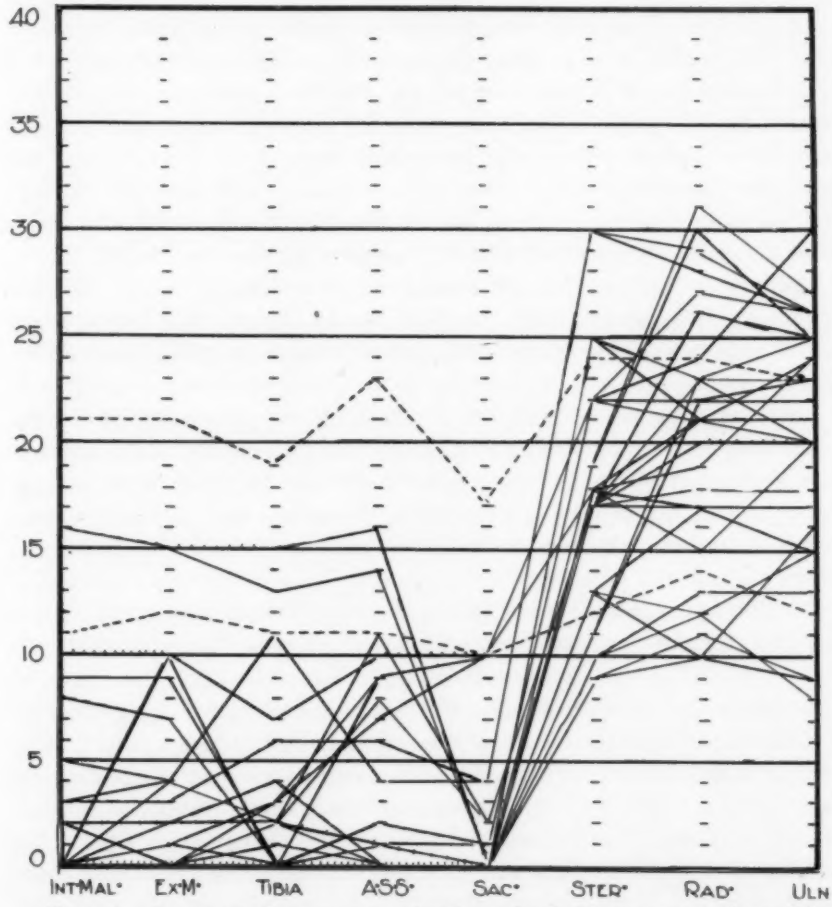

Chart 4.—Curve of vibratory sensation in author's fifteen cases of pernicious anemia.

has presented itself. Other patients examined in both exacerbation and remission have failed to show any differences in the type of curve obtained. These tabetic curves can be considered exceptional. Of this series of seventeen cases, there were definite neurologic findings in eight, suggestive findings in four, and five were neurologically negative.

In other neurologic conditions the findings vary. In syphilis of the central nervous system other than tabes the findings are not as constant

or as striking and in many instances may be normal, depending entirely of course on the part of the cord or brain involved and the extent of the lesion. In syphilitic meningomyelitis there may be impairment or absence of the vibratory sensation with a return to normal as improvement occurs. The findings in this condition often show marked asymmetry. Thus one patient had total flaccid paralysis of the right leg with loss of both superficial and deep sensibility corresponding to the level of the first lumbar segment. Spinal fluid and blood Wassermann tests were postive. The entire condition from onset to complete recovery covered a period of only three weeks. In vascular neurosyphilis, such as hemiplegia of thrombotic type, the vibratory sensation was found normal in two cases and impaired over the anterior sacral spines in one case. One patient with spastic paraplegia showed an absence of vibratory sensation over the anterior sacral spines, with normal readings over all other points. In two cases of general paresis normal readings were obtained. Several patients with general paresis also had the physical findings of early tabes with the characteristic tabetic curve. Of four patients with postencephalitic parkinsonian syndrome, one was slightly below normal over the lower extremities. The others were all within normal limits.

In other lesions of the cord, the findings naturally depend on the nature and location of the lesion with reference to the posterior columns. Five patients with transverse myelitis, in four of which it was traumatic, and in the fifth of the ascending infectious type, showed a total loss of the vibratory sensation below the level of the lesion with normal findings above the level.

Williamson,[10] whose work has been entirely qualitative, pointed out that the vibratory sensation could normally be felt over the anterior abdominal wall as well as over bony prominences and that in cases of cord tumor or other lesions in the region of the tenth, eleventh or twelfth dorsal segment, he found the vibratory sensation normal on the anterior abdominal wall above the level corresponding to the lesion and absent below that level. In one case of transverse myelitis, a gunshot wound involving the eleventh and twelfth dorsal segments, I was able to demonstrate this finding satisfactorily. In patients who are not too obese or in whom the abdominal wall is not too relaxed, the vibratory sensation can normally be demonstrated, although it is not felt as clearly, nor does it last as long as over the bony points.

In one patient with tumor of the cauda the vibratory sensation was absent over the anterior sacral spines and impaired over the sacrum, being normal elsewhere. The findings were of no value in diagnosis in this case. In disseminated sclerosis the peculiar patchwork pathology may give any variety of sensory findings. One patient showed total absence over the lower extremities, very high reading over the sternum and impaired sensation in the upper extremities. In multiple peripheral

neuritis, the vibratory sensation is uniformly impaired. In certain cases which closely resemble tabes in the early stages, the vibratory curve is of value in differential diagnosis. In diabetes mellitus with neurologic findings, the sensation usually shows the generalized impairment as found in any peripheral neuritis. One patient with gangrene of the right first toe showed impairment over the right lower extremity while the left side was above the usual normal.

SUMMARY

Examination of the vibratory sensation in one hundred patients showing no neurologic pathology gave findings which corresponded to the findings in one hundred similar cases reported by Wood. These normal curves have served as a satisfactory basis of comparison with the curves obtained in cases in which there are known pathologic changes of the central nervous system.

In tabes dorsalis all the patients examined showed an impaired vibratory sensation. Impairment or absence of the vibratory sensation over the sacrum may be the only neurologic sign of tabes. As the disease progresses, the vibratory sensation is gradually abolished in the lower extremities, while impairment in the upper extremities is rare. A characteristic curve presenting a sacral dip is therefore present in early tabes. The quantitative measurement of the vibratory sensation may be an important factor in the differential diagnosis of tabes from pernicious anemia and peripheral neuritis.

In other forms of syphilis of the central nervous system changes in vibratory sensation are not constant or characteristic.

In posterior lateral sclerosis a characteristic curve is also found showing a marked impairment in the earlier stages in the distal portions of the lower extremities with an approach to normal as the upper extremities are reached. As the disease progresses the curve approaches that of advanced tabes. In posterior lateral sclerosis, as in tabes, the impaired vibratory sensation may be the only neurologic finding.

In cord lesions the vibratory sensation may be of localizing value. In peripheral neuritis, there is a generalized impairment of the vibratory sensation, the upper and lower extremities being equally affected.

CONCLUSIONS

Tests for vibratory sensation if carefully performed may be of considerable value in diagnosis, calling the attention of the neurologist, in the absence of other findings, to an early posterior sclerosis.

A reasonable objection to the test is that it is time consuming. However, any method of quantitative determination of sensation which gives a large average of constant findings is to be welcomed in a field where heretofore only the presence or absence of the sensation could be determined.

LUMBAR PUNCTURE HEADACHE*

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Lumbar puncture headache is one of the most difficult and trying complications with which the practitioner of medicine has to cope. There seems to be no way to predict whether headache will follow lumbar puncture, and no method of prevention when such a complication is suspected. Patients are often told that lumbar puncture is simple and harmless, only to find that it is followed by a severe and stubborn headache which incapacitates them for days and weeks, and which seems to resist all known forms of relief. Not only is this problem of great practical importance, but from a scientific point of view it is of great interest, for the cause of lumbar puncture headache is probably closely linked with the hydrodynamics of the cerebrospinal fluid.

I shall present studies in a series of cases of lumbar puncture headaches, and suggest a method of treatment which has proved to be of distinct value in some of these cases.

REVIEW OF LITERATURE

Dana,[1] in 1917, stated that lumbar puncture headache was "more common in patients whose cerebrospinal fluid is negative, and in whom the fluid comes out under a low pressure." He concluded, moreover, that it made little difference in the production of a headache whether a large or a small amount of fluid was removed, or whether the fluid was removed slowly or rapidly. He ascribed the cause of the headache either to an irritation of the dural fibers of the fifth and occipital nerves caused by the removal of the water pad of the brain and leaving the brain to rest unprotected on the bone, or to an inhibition of the activity of the choroid plexus caused by the lumbar puncture. Rest in bed for three or four days he considered the best method of preventing a lumbar puncture headache.

Lott [2] studied the incidence of lumbar puncture headache in 700 cases in which lumbar puncture was performed, and concluded that the intensity and duration of the post-puncture reaction is in direct proportion to the degree of the intracranial pressure, patients with the higher

* From the Boston Psychopathic Hospital and the Philadelphia Orthopedic Hospital and Infirmary for Nervous Diseases.

1. Dana, C. L.: Lumbar Puncture Headache, J.A.M.A. **68**:1017 (April 7) 1917.

2. Lott, Y. C.: Observation and Technique of Lumbar Puncture in 700 Cases, M. Rec. **92**:717, 1917.

pressures developing the most marked reactions. His conclusions, however, were not based on manometric studies and therefore lost most of their value.

In 1918, MacRobert [3] presented the theory which seems to have been accepted widely. He said that lumbar puncture headache is due to two factors: first, an escape of fluid, and second, to a loss of the supporting cushion of the brain due to this loss of fluid. The dura is a thick, fibrous sac with no elastic or contractile tissue. Hence a hole here persists as a hole until it heals, which takes seven or eight days. Following a lumbar puncture the arachnoid, as it falls away from the needle, is swept snugly against the dura mater by the pressure of the fluid within the subarachnoid space. If the hole in the arachnoid does not approximate the hole in the dura, no fluid escapes, and no headache develops. If, however, the arachnoid clings to the needle as it is withdrawn, and is pulled back through the hole in the dura, fluid can escape by the approximation of these two holes, and lumbar puncture headache follows. The headache may be due to one of two causes: either to a loss of the supporting cushion of the brain thus causing the pressure of the brain against the bony surface of the skull, or by pressure of the brain against the basilar venous plexuses. In the latter case the blood which is about to leave the skull is impeded in its outflow, the resulting congestion causes a sudden rise of venous pressure which causes the headache, and which is relieved by lying down. In describing the course of events in the disappearance of the headache, MacRobert says that "in the course of a week the puncture hole heals, the fluid is rapidly made in sufficient quantity to fill and distend the entire sac, and the integrity of the brain cushion or water bed is reestablished. The headache, which was a purely mechanical affair dependent on the loss of that cushion, is gone."

When he suspended spinal cords and filled the dura with water, Greene [4] found that the large needles with a cutting edge in general use made large irregular holes in the dura, and thus permitted the escape of large amounts of fluid. He therefore used small bore needles with a round point and found that instead of cutting the dural fibers, such a needle merely separated them and was followed on withdrawal by an approximation of these fibers. In this way no fluid escaped and no headache ensued. He concluded that "the leakage is the result of the destruction of the longitudinal fibers composing the dural sac and the mutilation of the endothelial covering which necessarily follows the use

3. MacRobert, R. G.: The Cause of Lumbar Puncture Headache, J. A. M. A. **70**:1350 (May 11) 1918.

4. Greene, H. M.: A Technic to Reduce the Incidence of Headache Following Lumbar Puncture in Ambulatory Patients, Northwest Med. **22**:240, 1923.

of a large trocar or needle with a cutting edge." In 252 lumbar punctures with a needle with a round point and no cutting edge, he had an incidence of ten headaches, or 4 per cent. of all his cases.

Baruch [5] first attempted to prove experimentally the theory of MacRobert that fluid escapes through the hole in the dura after lumbar puncture. He performed lumbar puncture, and without withdrawing any fluid injected 3 c.c. of a 2 per cent. indigo-carmin solution into the subarachnoid space. He then plugged his needle with mandrin so that no fluid escaped. He then inserted a permanent catheter into the patient, in order to determine immediately the appearance of the dyestuff in the urine. In this experiment, with the lumbar puncture needle in situ, no dye appeared in the urine after sixty-three minutes. On withdrawal of the needle, however, the dye appeared in the urine in eight minutes. Baruch interpreted this as meaning that as long as the hole in the dura was blocked no fluid escaped and no dye appeared in the urine, but as soon as the hole in the dura became patent, fluid escaped and dye appeared in the urine. He took this as evidence of the fact that fluid escapes through the hole created in the dura by a lumbar puncture needle.

Stecher [6] continued these experiments, and attempted to determine whether more dye is excreted when the hole in the dura is open than when it is closed, and also whether it appears sooner under these conditions. He found that when the dura is open more dye appears than when it is closed, and concedes the fact that fluid drains off in this way. He points out, however, that the recovery of the dye in the urine does not give one a clue as to the amount of fluid actually lost, and remarks that if the loss of fluid by drainage were as great as the supporters of the theory believe, we should get much more serious symptoms than actually occur.

So much for the theory of the etiology of lumbar puncture headache. Definite efforts have been made to relieve this complication by medicinal treatment. Besides the use of liquids and coal tar derivatives which have been proved entirely useless, such drugs as pituitary extract and salines have been used. The latter have a much more rational physiologic basis than any of the other drugs. Baar [7] found that the incidence of headache after lumbar puncture was less when arsphenamin

5. Baruch, H.: Zur Ursache unangenehmer Nebenerscheinungen der Lumbaranesthenie zugleich ein Beitrag zur Funktionsprüfung des Liquorsystems, Berl. klin. Wchnschr. **38**:298, 1920.

6. Stecher, H.: Experimental Investigation of the So-Called "Stichlochdrainage" After Lumbar Puncture, Ztschr. f. d. ges. Neurol. u. Psychiat. **91**:114, 1924.

7. Baar, G.: New Procedure for the Prevention of Lumbar Puncture Headache, M. Rec. **98**:598, 1920.

was administered than when it was not given, and so he decided to give 0.5 per cent. saline intravenously immediately following lumbar puncture. Of ninety-three patients who received no saline after the puncture, fifty-one, or 55 per cent., developed headaches; of fifty patients who received saline, only three, or 6 per cent., developed headaches. Jacobaeus and Frumerie [8] observed that the pressure of the cerebrospinal fluid was almost zero in two patients with severe symptoms after lumbar puncture. Intraspinal injection of normal saline (35 to 90 c.c.) was followed by almost immediate recovery. The effects of hypotonic solutions in increasing the cerebrospinal fluid pressure has been demonstrated by Weed and McKibben,[9] Weed and Hughson [10] and others, and I shall attempt to demonstrate later how this action is of value in relieving lumbar puncture headaches.

Solomon [11] has used pituitary extract intramuscularly for the relief of lumbar puncture headache, and reports striking relief in certain cases. Moewes [12] advocated the use of epinephrin in lumbar puncture headaches and reported definite relief in a few cases. But while pituitary extract gives relief in certain cases, it is not successful in relieving all lumbar puncture headaches, nor is its physiologic action definite. It has been given because it is supposed to increase the cerebrospinal fluid pressure. Weed [13] found an increased flow in cerebrospinal fluid after injecting certain drugs and noting the outflow from a catheter which had been inserted into the aqueduct of Sylvius. Later, Cushing and Weed [14] demonstrated an increased flow of fluid following the injection of pituitary extract, and Halliburton and Dixon [15] have demonstrated an increased outflow after the injection of choroid plexus extract. Doubt has been thrown on these findings, however, by Becht

8. Jacobaeus, H., and Frumerie, M.: The Leakage of Spinal Fluid After Lumbar Puncture and Its Treatment, Acta med. Scandinav. **58**:102, 1923.

9. Weed, L. H., and McKibben, P. S.: Pressure Changes in the Cerebrospinal Fluid Following Intravenous Injection of Solutions of Various Concentrations, Am. J. Physiol. **48**:512, 1919.

10. Weed, L. H., and Hughson, W.: Systemic Effects of the Intravenous Injection of Solutions of Various Concentration with Special Reference to the Cerebrospinal Fluid, Am. J. Physiol. **58**:53, 1921.

11. Solomon, H. C.: Raising Spinal Fluid Pressure with Especial Regard to Its Effect on Lumbar Puncture Headache, J. A. M. A. **82**:1512 (May 10) 1924.

12. Moewes, M.: New Uses for Epinephrin in Clinic and Practice, Deutsch. med. Wchnschr. **50**:239, 1924.

13. Weed, L. H.: The Cerebrospinal Fluid, Physiol. Rec. **2**:171, 1922.

14. Weed, L. H., and Cushing, H.: Studies on the Cerebrospinal Fluid: The Effect of Pituitary Extract on Its Secretion, Am. J. Physiol. **36**:77, 1915.

15. Halliburton, W. D., and Dixon, W. E.: Secretion of the Cerebrospinal Fluid, J. Physiol. **47**:215, 1913.

and Gunnar.[16] These investigators, by means of an apparatus arranged so that volume changes could be detected in the spinal fluid, concluded that no increase in the production of fluid occurs with the administration of epinephrin, pituitary extract, pilocarpin or atropin, but that what occurs is a displacement of preformed fluid due to increased venous pressure. Hence the increase in pressure which seems to occur after the administration of these drugs is not due to an increased production of cerebrospinal fluid, and any therapeutic effect should therefore be transitory.

EXPERIMENTAL EVIDENCE

The incidence of lumbar puncture headache in only a limited percentage of cases, its apparent lack of connection with the amount of fluid withdrawn and with the rate of withdrawal, and its absence in certain diseases, made a predisposing factor seem likely. This factor we sought in the spinal fluid pressure. No attempt has been made to establish a theory of lumbar puncture headache; but a serious attempt has been made to show that pressure relations have a definite relation to the incidence of headache following lumbar puncture.

Ninety-one observations were made on patients at the Boston Psychopathic Hospital. Lumbar puncture was performed in the usual manner: the spinal fluid pressure was taken before any fluid was withdrawn, from 15 c.c. to 25 c.c. of spinal fluid was removed, and the pressure was again taken. The needle was then removed, and the patient was observed for from ten to fourteen days for the occurrence of a lumbar puncture headache. Large amounts of fluid were removed in order to determine the effect in the production of headache. Of the ninety-one cases, sixteen, or 17.5 per cent., developed headache, while seventy-five, or 82.5 per cent., did not develop headache. The amount of fluid withdrawn seemed to have no relation to the incidence of headache, for most of the patients who did not develop headache were relieved of 25 c.c. of fluid. Those who developed headache had from 10 to 25 c.c. of fluid removed. No direct relationship between the amount of fluid withdrawn and the incidence of headache could be demonstrated.

Sixteen of the ninety-one patients developed headache. All except one had an initial pressure of 100 to 160 mm. of water, with an average pressure of about 140 mm. In one case, the initial pressure was 55 mm. The outstanding feature in the cases in which there was headache, however, was the marked fall in the spinal fluid pressure after the withdrawal of fluid. In one case the pressure fell from an

16. Becht, F. E., and Gunnar, H.: A Study of the Volume Changes of the Cerebrospinal Fluid After Adrenalin, Pituitrin, Pilocarpine, and Atropine, Am. J. Physiol. **56**:231, 1921.

initial pressure of 55 to a pressure of 0. after 10 c.c. of fluid had been withdrawn. In another case the withdrawal of 25 c.c. of fluid resulted in the fall of the pressure from 140 mm. to 0. In all the other cases in which headache developed the withdrawal of fluid, whether in small or large amounts, resulted in a marked fall in spinal fluid pressure, varying from 100 to 140 mm. of water. It is likely that after the withdrawal of the needle the pressure continued to fall, probably reaching zero in some cases In one case of lumbar puncture headache, a second puncture revealed a spinal fluid pressure of zero. In any event, in those cases in which a post-puncture reaction developed there was a marked fall in the spinal fluid pressure after lumbar puncture and withdrawal of fluid. It therefore seemed rational to treat these patients by an attempt to increase the spinal fluid pressure. This was done by the use of pituitrin given intramuscularly and a hypotonic salt solution given intravenously.

If any definite conclusions can be drawn from these observations, it is not that in cases with a low initial pressure post-puncture headache is likely to develop, but rather that in cases with a marked disturbance in pressure relations as indicated by a marked fall in the spinal fluid pressure, a reaction will probably develop. This does not mean that a marked fall in pressure is the cause of the lumbar puncture headache. This probably is only a predisposing factor, and the leakage is probably the factor in maintaining the headache.

TREATMENT OF LUMBAR PUNCTURE HEADACHE

Drugs.—Pituitary extract was given to twelve of the sixteen patients who had developed lumbar puncture headache. In two of these patients no relief was obtained. In ten, however, the headache was relieved permanently. There was almost immediate relief in all cases. Further use of this drug at the Philadelphia Orthopedic Hospital has convinced us that in the less severe post-puncture reactions, pituitary medication is of distinct benefit in giving relief, but that in severe cases of lumbar puncture headache it often has no effect. In the two cases in which there was a pressure of zero hypotonic saline was given intravenously. In one case the headache disappeared after three hours, and in the other after two hours. In the severe cases of lumbar puncture headache, the use of hypotonic saline is more beneficial than pituitary extract, and the effect on the spinal fluid pressure is much more lasting. Weed and Hughson state that the effect of hypotonic saline on the spinal fluid pressure is both prompt and enduring. Intravenous injection of the saline may be uncomfortable, but it is less so than the headache; and it brings relief. Usually about 100 c.c. of a 0.5 per cent. solution of sodium chlorid is a sufficient dose.

Needle.—The size of the needle is probably an important factor in the determination of the size of the hole in the dura, and therefore the amount of leakage. A fine needle should be used. Recently we have been using a very fine needle, and in twelve lumbar punctures with these needles no headaches have resulted.

Rest.—Rest in bed is an important factor. Patients should be kept in bed at least twenty-four hours, and if possible, forty-eight hours. We have seen patients who have been able to get up and walk around after a few hours without developing a headache. They probably would not have developed a headache under any circumstances. For the majority of patients, rest in bed with the head low should be insisted on.

Diet.—So far as we have been able to observe, the performance of lumbar puncture on fasting patients is not an important factor in preventing headaches.

CONCLUSIONS

1. Patients who show a marked fall in spinal fluid pressure after lumbar puncture are prone to develop lumbar puncture headache.

2. Pituitary extract (1 c.c. intramuscularly) is of great benefit in most cases of lumbar puncture headache.

3. Hypotonic saline (100 c.c. of 0.5 per cent.) is of value in severe and persistent cases of post-puncture headache.

SPINA BIFIDA OCCULTA CERVICALIS

REPORT OF A CASE *

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CHICAGO

Hypertrichosis over a dimpled area in any region of the vertebral column is suggestive of spina bifida occulta. It is considered almost pathognomonic for those types of spina bifida in which a protruding cystic tumor is absent. The hairy area usually covers a defect in the vertebra in the form of an absent or split spinous process, a cleft in the arch of the vertebra, or, as Beck [1] asserts, even a mere depression in the spinous process.

Some even go so far as to include with spina bifida occulta conditions in which the anomaly is not in the bony structures, but presumably in the spinal cord itself. On purely clinical grounds, Fuchs [2] has described such conditions as myelodysplasia. He considers spina bifida occulta as the most severe form or stage of what he terms myelodysplasia. Few cases of the latter are mentioned in the literature. Spiller [3] recently has described one, and Woltman [4] mentions three instances in his statistics comprising 187 cases of spina bifida. Whether myelodysplasia of Fuchs belongs to the same group of disorders as spina bifida occulta or is something else, the fact remains that in spina bifida skin or bony lesions may be insignificant, while spinal cord disturbances may be pronounced and dominate the entire clinical picture. The variety of spinal cord symptoms is great, depending on the localization of the spina bifida and concomitant pathologic conditions—tumors, connective tissue bands, and adhesions between the skin and the spinal membranes pressing on the underlying cord. The evidences of disturbances of the spinal cord may be flaccid or spastic paralysis,

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* From the Neurological Service of Cook County Hospital, Chicago.

* Read before the Chicago Neurological Society, Feb. 19, 1925.

1. Beck, Otto: Spina bifida occulta und ihre ätiologische Beziehung zu Deformitäten der unteren Extremität, Ergebn. d. Chir. u. Orthopädie **15**:493, 1922.

2. Fuchs, A.: Ueber den klinischen Nachweis kongenitaler Defektbildungen in den unteren Rückenmarksabschnitten, Wien. med. Wchnschr. **59**:2142, 1909.

3. Spiller, W.: Congenital and Acquired Enuresis from Spinal Lesion. (a) Myelodysplasia; (b) Stretching of the Cauda Equina, Am. J. M. Sc. **151**:469, 1916.

4. Woltman, H. W.: Spina Bifida. A Review of 187 Cases Including Three Associated Cases of Myelodysplasia Without Demonstrable Bony Effect, Minnesota Med. **4**:244, 1921.

paralytic club foot, urinary or rectal incontinence (Katzenstein,[5] Delbet[6]), neuralgic pains (Beck[1]), sensory disturbances, muscular atrophies with fibrillary twitchings, trophic disturbances (Jones,[7] Curtius,[8] Elsberg,[9] Tutyschkin,[10] Durand[11]), etc. Of the foregoing conditions, the most unusual is a progressively developing spastic paralysis in the upper and lower extremities, the sensory disturbances being exceedingly mild or absent.

REPORT OF A CASE

History.—A man, aged 36, was admitted to my service at the Cook County Hospital, Oct. 10, 1924, complaining of weakness in the upper extremites. He left the hospital after a stay of three days, and returned Jan. 15, 1925, with the same complaints. He had been in comfortable health until about May, 1922, when he noticed "stiffness" in the fingers of the right hand. Two months later, the right arm, and twelve months after the onset of the disease, the left arm, became stiff. Eight months before admission, stiffness set in in the right leg, and "the toes would catch the floor"; this was followed two months later by a similar trouble in the left leg.

His mother had told him that in early childhood a tumor had been removed from his neck, and in 1904 he had had an attack of typhoid fever. In spite of the fact that he was a heavy smoker and drinker, he always enjoyed good health, was able to attend to his work as a team driver, had no difficulty in walking or using his upper extremities, had no gastro-intestinal, genito-urinary, cardiovascular or venereal diseases.

Examination.—The man was well nourished; his head was somewhat large and slightly tilted toward the right side; a moderate scoliosis was present in the mid-dorsal region. In the lower cervical region, at the level of the sixth cervical vertebra, there was a slightly reddened, hairy area, the size of a dime, containing a small depressed scar in the center. Palpation of this area provoked a "queer" sensation and revealed underneath a slight depression in the place of the spinous process. In walking, the patient kept his body slightly bent forward, the arms hanging extended, the forearms pronated, the wrist extended and the terminal phalanges flexed. The upper extremities, especially the right forearm, appeared atrophied, but no atrophy was present in the small muscles of the hands (thenar, hypothenar and interosseal). Some atrophy was noticeable in the scapular muscles, especially in the infraspinati, which occasionally

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5. Katzenstein, M.: Beitrag zur. Pathologie und Therapie d. Spina bifida occulta, Arch. f. klin. Chir. **64**:607, 1901.

6. Delbet, P., and Léri, A.: Incontinence d'urine dite essentielle. Spina bifida occulta. Operation. Guérison, Bull. de l'Acad. de méd. **89**:49, 1923.

7. Jones: Spina bifida occulta, Brit. M. J., Jan. 24, 1891, p. 173.

8. Curtius: Beitrag z. Pathologie d. Spina bifida lumbo-sacralis, Arch. f. klin. Chir. **45**:194, 1892.

9. Elsberg: Spina Bifida Occulta, with Thoracic Disturbances Followed by Fibro-Lipoma of Cauda Equina. Operation. Recovery, J. Nerv. & Ment. Dis. **38**:289, 1911.

10. Tutyschkin, P.: Ein seltener Fall von Spina bifida mit späterem Einsetzen der Symptome seitens Cauda equina, Conus medullaris, Epiconus Minori, etc., Neurol. Centralbl. **33**:153, 220, 1914.

11. Durand: Spina bifida occulta, Lyon méd. **132**:246, 1923.

showed fibrillary twitchings. Movements, active and passive, were freely carried out in the lower extremities, and the patient was able to move about, although the gait was markedly spastic. In the knees and ankles, passive movements met with some resistance, but they were free in the hip joints. In the upper extremities, active movements in the shoulders and elbows were entirely absent, while passive movements in these joints met with great resistance. In the elbows, the resistance to passive movements was less marked, while in the wrists and fingers there was no resistance. Active movements of these parts were performed with great ease and freedom; for instance, in spite of their clawlike position, the fingers could be extended, flexed and abducted and exhibited good power, especially on the left side. The tendon reflexes, except that of the biceps bilaterally, were markedly exaggerated, with bilateral ankle clonus and Babinski sign; the abdominal and cremasteric reflexes were absent.

The cranial nerves, speech and mentality showed no abnormalities; the pupils reacted normally to light and in accommodation. The tongue protruded normally and showed no fibrillary twitchings or atrophy, but occasionally exhibited coarse tremor. Electrical tests revealed normal responses of the nerves and muscles to both the faradic and galvanic currents. Sensibility was normal, except for loss of the stereognostic sense bilaterally, and loss of touch sense in both thumbs and of the position sense in the fingers of the left hand. The visceral organs—the heart, lungs, kidneys—were normal. The urine contained neither sugar nor albumin. The blood pressure was 175 systolic and 120 diastolic. The Wassermann test of both the blood and the spinal fluid was negative. Roentgen-ray examination revealed a small cleft in the spinous process of the sixth cervical vertebra.

Summary.—The findings in this case were: gradual development of a motor spastic disturbance in all four, but especially in the upper, extremities in the presence of a peculiar circumscribed skin lesion in the lower cervical region and associated with mild sensory disturbances in the fingers of both hands. The motor disturbances, in contrast, affected particularly the proximal portions of the upper extremities (shoulders and elbows).

I found only three cases of spastic paralysis recorded, those of Reiner,[12] Henneberg,[13] and Marie and Léri.[14] Reiner's patient, aged 7, began to develop spastic paralysis at the age of 2; Henneberg's patient, aged 32, from childhood, had difficulty in walking, while Marie and Léri's patient, aged 54, began to show spastic symptoms at the age of 46. The localizations of the spina bifida were: in Reiner's case, in the dorsal region, in Henneberg's case, in the lumbar region; only in Marie-Léri's case did it occupy the cervical region—over the seventh cervical vertebra. The clinical picture in Marie-Léri's patient strikingly resembled that in mine. There was weakness and rigidity, at the age

12. Reiner, M.: Ueber einen Fall von Spina bifida occulta dorsalis, Wien. klin. Rundschau **15**:325, 1901.

13. Henneberg: Spina bifida occulta mit spastischer Parese der Beine, Berl. klin. Wchnschr. **58**:1235, 1921.

14. Marie, Pierre, and Léri, A.: Spina bifida occulta cervical révélée exclusivement par une quadriplégie à debut extrémement tardif (à quarante-six ans), Bull. et mém. Soc. méd. d. hôp. de Paris **46**:1138, 1922.

of 46, in the left upper extremity; four years later, the same condition developed in the left lower extremity, followed two years afterward by involvement of the right upper extremity. At the time of the examination (eight years after the onset of the rigidity), complete quadriplegia was present, with practically no sensory, genito-urinary or other disturbances. The tendon reflexes were exaggerated, except the triceps and the radial reflexes, which were absent; while cutaneous signs of spinal bifida, such as hypertrichosis, pigmentation, dimpling and telangiectasis, were entirely absent. However, a roentgen-ray examination revealed a defect in the arch of the seventh cervical vertebra and its spinous process. A tumor was looked for during an operation, but was not found in the vertebral canal outside the dura (which was not opened), and Marie and Léri believe that the tumor was intramedullary.

Aside from the rarity of spastic phenomena as the principal if not the only manifestation of spina bifida, the localization of this condition in the cervical region is rare. I found only eight cases recorded: Milner,[15] two; Sever,[16] four; Clark,[17] one, and Marie-Léri, one. In all these cases, as well as in mine, the spina bifida was located in the lower half of the cervical region, although Feil[18] points out that it may also occur in the upper cervical region but remain unrecognized. The clinical picture of upper cervical bifida, according to Feil, is characteristic: the neck is very short or absent, the hair line reaches the dorsal vertebrae, and the movements of the head are greatly limited. The condition much resembles that described by him and Klippel[19] in a man in whom the cervical portion of the vertebral column was absent, that is, had failed to develop.

There can be no doubt that many cases of spina bifida occulta are not recognized, especially when the external, cutaneous signs are absent. Statistics, therefore, show that the cystic type of spina bifida is more common than the occulta variety. Thus Voltman found, among 187 cases recorded in the literature, 138 instances of the former and forty-six (24.6 per cent.) of the latter; this he admits is a low figure, for many persons, he states, "carry about spina bifida occulta without ill effects." This was true, for instance, in the cases of Reiner, Henneberg and Curtius, in which the disturbances were slight; they were entirely absent in the cases of Milner and Marie-Léri and in mine. The operation

15. Milner: Spina bifida occulta, Deutsch. med. Wchnschr. **31**:1579, 1905.

16. Sever, J. W.: Spina bifida occulta, Boston M. & S. J. **161**:388, 1909.

17. Clark, S. N.: Report of a Case of Spina Bifida Occulta in the Cervical Region, J. Nerv. & Ment. Dis. **48**:201, 1918.

18. Feil, A.: Sur la localization du spina bifida dans la région cervicale, Progrès méd. **36**:510, 1920.

19. Klippel, M., and Feil, A.: Un cas d'absence des vertèbres cervicales, Nouv. icon. de la Salpètrière **25**:223, 1912.

on the neck which my patient underwent in early childhood was evidently for a cystic spina bifida which had healed and caused him no trouble until late in life. The nature of the trouble that flares up causing a progressive spinal cord lesion is to be sought in adhesions or tumors, which when recognized and removed may lead to recovery (Elsberg's case,[9] for instance). Unfortunately, in many cases of spina bifida occulta the roentgen-ray examination is not satisfactory, and may prove negative because of ossification of the membrana reuniens, a connective tissue membrane covering the gap in the vertebral arches or processes.

One point should be emphasized. It was found, especially by Recklinghausen,[20] that in spina bifida the spinal cord is often elongated, in some cases reaching the second sacral segment. Such cords, of course, have no cauda equina, and if a spinal puncture is made in the usual region, below the first or second lumbar vertebra, one is liable to injure the spinal cord. Therefore, Beck advises caution in making spinal punctures in patients in whom a spina bifida occulta is suspected.

CONCLUSIONS

1. Various spinal cord signs and symptoms may be caused by spina bifida occulta.

2. Spina bifida occulta may remain latent causing no symptoms.

3. As external, cutaneous signs of spina bifida may be absent or very mild, one should consider this condition in some cases of paralytic, sensory, genito-urinary, trophic and other disturbances with obscure etiology.

DISCUSSION

DR. D. M. OLKON: Were there changes in temperature sensibility?

DR. HASSIN: No.

DR. PETER BASSOE: Two cases of spina bifida occulta have been reported before this society at previous meetings. One, of the cervical spine was reported by Dr. Clark.[21] There was marked hyperextensibility and clumsiness of the fingers in that case. When first examined at the clinic, a little dimple with hair in it was found at the back of the neck and a roentgenogram showed a cleft of the arch of the third cervical vertebra.

The second case, involving the lumbar region, was reported by myself,[22] and illustrates the tendency to development of severe symptoms late in life. A man, had had a pes cavus since birth; at about the age of 50 he developed marked urinary incontinence, and then spasticity with numbness of the feet and extensive thermanesthesia and analgesia; a cleft in the arch of the fifth lumbar vertebra and a hairy dimple over it clinched the diagnosis. The patient died from urinary sepsis. I showed the specimen here. It revealed an enlargement

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20. Recklinghausen, F.: Untersuchungen über die spina bifida, Virchow's Arch. f. path. Anat. **105**:243, 1886.

21. Clark, Samuel N.: J. Nerv. & Ment. Dis. **48**:201, 1918.

22. Bassoe, P: Internat. Clin., 1922.

of the conus, due to a congenital lipoma with an enormous number of medullary fibers running through the fatty masses. Evidently, the tumor process had started trouble in the disordered nervous tissue late in life.

DR. JULIUS GRINKER: In cases like the one reported by Dr. Hassin in which the disease is evidenced late in life, would it not be wise to remove the tumor with the view of improving the condition?

DR. HASSIN: The cases mentioned by Dr. Bassoe support the view that spina bifida occulta may be the cause of spinal cord disturbances evidenced by such disturbances as urinary difficulties and trophic skin lesions. Cases similar to the second one he mentioned—congenital lesions in the conus associated with lipomatous growths—have been found by surgeons and accidentally by pathologists. An operation on the man here presented is strongly indicated; unfortunately, the patient refuses to consent. Marie and Léri used roentgen-ray therapy for a time, but as improvement was insignificant an operation was performed. No tumor was found, and it was decided that it must be intramedullary.

CORRECTION

In the ARCHIVES OF NEUROLOGY AND PSYCHIATRY, October, page 538, appeared an abstract of an article published in *Brain*, entitled "A Clinical Study of Increased Intracranial Pressure in Sixty Cases of Cerebral Tumor." The name of the author, Dr. W. Russell Brain, was incorrectly given as W. Russell. The reason for the confusion which resulted in the elimination of the last name of the author is obvious.

BASAL METABOLISM IN MENTAL DISEASE

K. M. BOWMAN, M.D., AND C. C. FRY, M.D.
BOSTON

Recently, a number of studies of basal metabolism in mental disease have been published. These studies have been undertaken mainly with the endeavor to clear up, if possible, some of the disputed points regarding so-called functional psychoses. Recent studies of the endocrine glands and their effect on the emotional life of the individual have caused many to wonder whether or not such studies will serve to explain some mental disorders concerning which there is so much controversy. The study of the basal metabolism is considered by many to be a test of thyroid activity. It is well to point out that basal metabolism is affected not only by the thyroid gland but by other glands of internal secretion, such as the gonads, the suprarenals and the pituitary, and that there are many other factors controlling basal metabolism which are but little understood. Sleep produces a lowering of metabolism. The question of a cerebral control of metabolism has been raised by some investigators. It should also be emphasized that practically all errors of technic and lack of cooperation by the patient give increased readings.

Various disorders of metabolism have been reported in mental disease. Kraepelin [1] summarizes the physical findings in dementia praecox, and points out that Grafe found a distinct slowing of metabolism in catatonic stupor with a lowering of heat production to 39 per cent. of normal, and that Bornstein reported "a considerable lowering of the need of oxygen which cannot be removed by thyroidin."

In 1922, one of us [2] reported a tendency toward low basal metabolism in dementia praecox, seven of ten cases showing abnormally low basal metabolism. In a second article,[3] the basal metabolism was reported in fifty cases of mental disease of all types. In forty-two of these cases there were minus readings, and in twenty-seven the basal metabolism was lower than — 10 per cent., which is commonly considered the lower limit of the normal.

Gibbs and Lemcke [4] reported a series of eleven cases of dementia praecox and fifteen cases of manic-depressive psychosis. In a large number of their cases there were normal readings, but there was a definite tendency toward low readings.

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1. Kraepelin, E.: Text Book of Psychiatry, Livingstone, Vol. 3, Pt. 2, 1919.
2. Bowman, K. M.; Eidson, J. P., and Burladge, S. P.: Biochemical Studies in Ten Cases of Dementia Praecox, Boston M. & S. J. **187**:358 (Sept. 7) 1922.
3. Bowman, K. M., and Grabfield, G. P.: Basal Metabolism in Mental Disease, Arch. Neurol. & Psychiat. **9**:358 (March) 1923.
4. Gibbs, C. E., and Lemcke, D.: Study of Basal Metabolism in Dementia Praecox and Manic Depressive Psychoses, Arch. Int. Med. **31**:102 (Jan.) 1923.

TABLE 1.—*Basal Metabolism in Author's Cases*

Case No.	Age	Sex *	Basal Metabolism	Diagnosis
1	64	♀	—14	Senile psychosis
2	73	♂	+1	Psychosis with cerebral arteriosclerosis
3	42	♂	—25	General paresis
4	45	♂	+10	General paresis
5	24	♀	—8	Psychoses with other brain and nervous diseases (disseminated sclerosis)
6	19	♀	—5	Lethargic encephalitis
7	17	♀	—6	Lethargic encephalitis
8	18	♀	—8	Lethargic encephalitis
9	24	♂	—2	Lethargic encephalitis
10	34	♂	—30	Lethargic encephalitis
11	35	♂	—4	Alcoholic psychosis
12	22	♂	—5	Alcoholic psychosis
13	36	♂	Normal	Psychosis with pellagra
14	43	♀	+12	Psychosis with pellagra
15	51	♀	—11	Psychosis with other somatic diseases (delirium of unknown origin)
16	26	♀	+45	Delirium of unknown origin
17	27	♀	—20	Cardiorenal type
18	48	♀	—2	Acromology
19	23	♂	—5	Manic depressive (manic)
20	41	♂	+28	Manic
21	36	♀	+9	Manic
22	38	♀	—3	Manic
23	54	♂	—27	Depression
24	28	♀	+4	Depression
25	36	♀	—12	Depression
26	33	♀	+11	Depression
27	23	♂	—0	Depression
28	18	♀	+6	Depression
29	28	♀	+13	Depression
30	28	♀	+6	Depression
31	35	♀	—2	Depression
32	40	♂	—7	Depression
33	29	♂	Normal	Depression
34	32	♀	—15	Depression
35	25	♂	Normal	Depression
36	30	♀	—21	Depression
37	31	♀	+7	Depression
38	22	♂	—16	Depression
39	35	♀	—8	Depression
40	19	♀	17	Hypomanic
41	26	♀	—10	Hypomanic
42	25	♀	—6	Hypomanic
43	19	♂	—16	Hypomanic
44	31	♀	+11	Dementia praecox
45	39	♀	+2	Dementia praecox
46	19	♀	—6	Dementia praecox
47	41	♀	—4	Dementia praecox
48	17	♀	—11	Dementia praecox
49	20	♀	+7	Dementia praecox
50	22	♂	—7	Dementia praecox
51	37	♀	+9	Dementia praecox
52	25	♀	—2	Dementia praecox
53	39	♀	+7	Dementia praecox
54	22	♀	—25	Dementia praecox
55	15	♂	—13	Dementia praecox
56	19	♂	—17	Dementia praecox
57	19	♂	—20	Dementia praecox
58	33	♂	—5	Dementia praecox
59	28	♂	—4	Dementia praecox
60	24	♂	+34	Dementia praecox
61	16	♂	—7	Dementia praecox
62	29	♀	—10	Dementia praecox
63	21	♂	—2	Dementia praecox
64	33	♀	—30	Dementia praecox
65	22	♀	—5	Dementia praecox
66	15	♀	—17	Dementia praecox
67	24	♂	—18	Dementia praecox
68	40	♀	—6	Paranoid condition
69	36	♀	—5	Paranoid condition
70	56	♂	—11	Paranoid condition
71	57	♂	+48	Paranoid condition
72	29	♂	—2	Paranoid condition
73	34	♂	—9	Paranoid conditoin
74	27	♂	—29	Paranoia
75	18	♂	—0	Epileptic psychosis
76	35	♂	—10	Epileptic psychosis
77	18	♂	—5	Epileptic psychosis
78	12	♂	—5	Epileptic psychosis
79	20	♂	—8	Epileptic psychosis
80	38	♀	+8	Psychoneurosis
81	38	♀	—5	Psychoneurosis

* In this column, ♂ indicates male, ♀ female.

TABLE 1.—*Basal Metabolism in Author's Cases—Continued*

Case No.	Age	Sex *	Basal Metabolism	Diagnosis
82	33	♀	+3	Psychoneurosis
83	21	♀	+7	Psychoneurosis
84	28	♀	−9	Psychoneurosis
85	36	♂	−9	Hysteria
86	26	♀	+32	Hysteria
87	20	♀	+2	Hysteria
88	21	♀	+5	Hysteria
89	29	♀	−18	Hysteria
90	57	♀	−7	Psychasthenia
91	31	♀	+2	Psychasthenia
92	38	♀	−9	Psychasthenia
93	21	♀	−4	Psychasthenia
94	34	♂	+7	Anxiety type
95	17	♂	+21	Anxiety type
96	45	♀	Normal	Anxiety type
97	22	♀	+3	Psychosis with psychopathic personality
98	19	♀	−13	Psychosis with psychopathic personality
99	15	♂	−18	Psychosis with psychopathic personality
100	43	♀	+31	Psychosis with psychopathic personality
101	22	♀	−14	Psychosis with psychopathic personality
102	56	♀	−11	Psychosis with psychopathic personality
103	17	♂	Normal	Psychosis with psychopathic personality
104	31	♀	+15	Psychosis with psychopathic personality
105	29	♀	−5	Psychosis with mental deficiency
106	24	♀	+11	Psychosis with mental deficiency
107	16	♀	+18	Psychosis with mental deficiency
108	21	♀	+17	Psychosis with mental deficiency
109	30	♀	+13	Psychosis with mental deficiency
110	19	♀	−9	Psychosis with mental deficiency
111	37	♀	+30	Undiagnosed psychosis
112	23	♀	−8	Undiagnosed psychosis
113	24	♀	+1	Undiagnosed psychosis
114	31	♀	+10	Undiagnosed psychosis
115	20	♂	−1	Undiagnosed psychosis
116	27	♀	−12	Undiagnosed psychosis
117	25	♂	−6	Undiagnosed psychosis
118	34	♀	−9	Undiagnosed psychosis
119	14	♀	−10	Without psychosis (conduct disorder)
120	9	♂	Normal	Conduct disorder
121	18	♀	+40	Without psychosis (for diagnosis)
122	21	♀	−17	For diagnosis
123	24	♀	+8	For diagnosis
124	18	♀	+7	For diagnosis
125	24	♀	+3	For diagnosis
126	19	♂	−9	For diagnosis
127	12	♂	+9	Epilepsy—Petit mal
128	9	♀	−7	Epilepsy—Petit mal
129	20	♀	−2	Epilepsy
130	24	♂	−7	Epilepsy
131	18	♀	+7	Epilepsy
132	8	♀	−11	Epilepsy
133	20	♀	+5	Epilepsy
	38	♂	−2	Epilepsy
134	26	♂	Normal	Epilepsy
135	19	♂	−2	Epilepsy
136	19	♀	+18	Mental deficiency
137	17	♀	+8	Mental deficiency
138	27	♀	+1	Mental deficiency
139	17	♀	+10	Mental deficiency
140	15	♀	−14	Mental deficiency
141	19	♀	+8	Mental deficiency
142	15	♂	+7	Mental deficiency
143	23	♂	−16	Psychopathic personality
144	21	♂	−10	Psychopathic personality
145	11	♀	−16	Psychopathic personality
146	16	♀	−11	Psychopathic personality
147	17	♂	−1	Psychopathic personality
148	27	♀	−5	Psychopathic personality
149	9	♂	−4	Psychopathic personality
150	18	♂	−8	Psychopathic personality
151	17	♂	+32	Psychopathic personality
152	20	♀	+1	Psychopathic personality
153	24	♀	−1	Psychopathic personality
154	18	♀	+4	Psychopathic personality
155	15	♀	−13	Psychopathic personality
156	16	♀	+5	Psychopathic personality
157	18	♀	−15	Psychopathic personality
158	20	♀	−15	Psychopathic personality
159	18	♂	−6	Psychopathic personality
160	21	♂	−10	Psychopathic personality
161	17	♀	−13	Psychopathic personality

* In this column, ♂ indicates male, ♀ female.

Farr [5] has reported the results of the examination in 100 cases. He found 28 per cent. of his cases were below normal and 13 per cent. were above normal, making a total of 41 per cent. of his cases which showed abnormal basal metabolic rates. In dementia praecox, 37 per cent. of his cases were below normal and 17 per cent. were above normal, so that more than 50 per cent. of his cases of dementia praecox showed abnormal basal metabolic rates. In cases of mood disorders, 24 per cent. were below normal and 8 per cent. above.

Walker [6] made 200 estimations in forty-four cases of mental disorders. Thirty of these were cases of dementia praecox. He found that in 50 per cent. of the latter cases the basal metabolism was lower than — 10 per cent. In the other 50 per cent. the rate was either normal or slightly in excess. The basal metabolism in the subnormal cases ranged from — 12 per cent. to — 47 per cent., averaging — 20 per cent. All except two of his other cases fell within normal limits.

Talbot, Hendry and Moriarty [7] reported the basal metabolism in eleven children who had idiopathic epilepsy. The results showed that the rate was normal or elevated in all instances.

Boothby and Sandiford [8] reported twenty-eight cases of mental disease, and found the basal metabolism to be below — 10 per cent. in two cases and above + 10 per cent. in two cases. Studies were also made in a number of allied conditions . . . forty-two cases of migraine, 191 cases of "chronic nervous exhaustion," 210 cases of "neurasthenia," twenty-two cases of "cardiac neurosis," twenty-four cases of epilepsy and six cases of encephalitis. The vast majority of their readings in all of these conditions were within normal limits, and there was no striking evidence of any tendency toward either a decreased or increased basal metabolism in any of the groups mentioned.

Whitehorn [9] reported a series of cases of manic-depressive psychosis and cases of dementia praecox. In seventeen cases of manic-depressive psychosis, fourteen were within the normal limits of plus and minus 10 per cent.; one was below, and two were above. In eleven cases of dementia praecox, three cases were within the normal limits of plus and minus 10 per cent., whereas seven cases were abnormally low, and one

5. Farr, C. B.: Results of Basal Metabolism Tests in One Hundred Mental Cases, Arch. Neurol. & Psychiat. **12**:518 (Nov.) 1924.

6. Walker, J.: Basal Metabolism in Mental Disorders, Especially Dementia Praecox, and the Influence of the Diathermic Current on the Same, J. Ment. Sc. **70**, No. 288, January, 1924.

7. Talbot, F. B.; Hendry, M., and Moriarty, M.: The Basal Metabolism of Children with Idiopathic Epilepsy, Am. J. Dis. Child. **28**:419 (Oct.) 1924.

8. Boothby, W. M., and Sandiford, I.: Basal Metabolism. Physiological Reviews, **4**, No. 1, January, 1924.

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case was abnormally high. Whitehorn also states that the diagnosis in the case with high basal metabolism was questionable, and it was perhaps not a true case of dementia praecox.

In the present series, 161 cases were studied. They are listed according to diagnosis. The important findings may be summarized as follows if plus and minus 10 per cent. are taken as the normal limits for basal metabolism:

In twenty-four cases of schizophrenia (dementia praecox), seven cases showed abnormally low readings. Eighteen of twenty-four cases showed minus readings. The manic-depressive group showed a tendency toward low readings in the depressive and hypomanic stages, but this tendency was not as pronounced as in the schizophrenic cases. The cases of psychosis with epilepsy and epilepsy without psychosis were mostly within normal limits, but there was a tendency toward minus readings rather than plus readings. In cases of psychosis with mental deficiency and mental deficiency without psychosis the cases were mostly

TABLE 2.—*Basal Metabolism in Schizophrenia*

	Cases	Low	Normal	High
Bowman, Eidson and Burladge	10	7	3	0
Bowman and Grabfield	5	4	1	0
Farr	35	12	17	6
Walker	30	15	13	2
Whitehorn	11	7	3	1*
Bowman and Fry	24	7	15	2
Total	125	52	52	11

* Diagnosis doubtful.

within normal limits but there was a definite tendency toward plus readings. Nineteen cases of psychopathic personality were studied. Seven of these showed abnormally low readings and two abnormally high readings. Fifteen of the nineteen cases showed minus readings.

It would, therefore, appear that in three important conditions in which the emotional life of the individual is primarily involved, namely, schizophrenia, manic-depressive psychosis and psychopathic personality, there is a definite tendency toward a low basal metabolism, and that very few cases show an increased basal metabolism.

From the standpoint of the relationship of the endocrine glands to the emotional life of the individual this appears to be an observation meriting further study.

Table 2 shows the basal metabolism in all the cases that could be collected from the literature on the subject. It points out in a striking way the tendency toward a low basal metabolism in schizophrenia (dementia praecox), which is consistently reported by every observer.

Boston Psychopathic Hospital.

Clinical and Occasional Notes

CONGENITAL TOTAL HEMIHYPERTROPHY

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NEW YORK

While the literature reveals forty-four instances of this anomaly, the two cases here described are the first examples reported of two in the same family.

The patients, a boy of 17 years and a girl of 12 years, brother and sister, came to the neuropsychiatric clinic of the Brooklyn Hospital. The girl was referred by the inspector of ungraded classes, Board of Education, City of New York, because of backwardness in school; her brother was brought by request, because the mother stated that he was subject to "fainting spells." The mother said that she had been aware of the hypertrophic condition since birth, but as it did not incapacitate the children beyond the necessity of wearing special shoes to overcome a slight limp, she never sought medical advice.

REPORT OF CASES

CASE 1.—S. L., a boy, aged 17 years, whose birth and infancy were normal, was "delicate" as a child. He had frequent colds and was unable to put on weight, but development was normal. No direct hereditary history of epilepsy was elicited. The father and mother, however, suffered from "nervousness," and his sister, whose case is the second one reported in this article, is mentally defective. His school record was that of the average child. He graduated from the public school at 14 years, and is at present attending Erasmus Hall High School.

At 12 years of age (1920), he was struck on the side of the head by a baseball, and since then had had grand mal attacks occurring on the average twice a week. He was put on alternate courses of phenolbarbital and caffein with marked success, so far as the attacks were concerned. He was difficult to handle and a little shy on the first examination, but his reserve wore off, and he began to manifest a good many of the characteristics of the public school boy. I observed a tendency to "look for trouble." He showed self-assertiveness and a general egocentric point of view, coupled with a tendency to depreciate the qualities of others in loud and often inelegant language. I also observed sullenness following even the slightest correction, and occasional moodiness, which is perhaps part of the epileptic picture. His weight was 110 pounds (49.9 kg.); his height, 61 inches. He was poorly developed and nourished. There was hypertrophy of the entire right side from head to foot. The measurements are presented in Table 1.

The boy was placed in the hands of a physical instructor to build up his physical condition. He has gained 17½ pounds (7.9 kg.) in eighteen months, has increased noticeably in muscular strength and activity, and his general health is excellent. A lacto-vegetable diet has been prescribed; also pheno-

barbital in doses of 0.25 gm. after each meal and before retiring. The boy has had but one convulsion in eighteen months, and this was due probably to carelessness in diet. I have also made an effort to keep the boy's mind occupied with review work in school, handiwork, sports and other activities. During this past summer (1924) he was sent to a boys' camp, where he took part in a water carnival and won several prizes, notably for good diving. He also played a good game of tennis for his age. He played baseball and basketball and learned a little about paddling a canoe. I found that he especially needed development in a sense of good sportsmanship and fair play. Prompt and cheerful obedience and helpfulness to others was stressed, with discouragement of boasting, teasing and slangy disparagement of others. The laboratory findings, including the blood and spinal fluid Wassermann tests, were normal.

His present measurements are noted in Table 2.

TABLE 1.—*Measurements of First Patient at First Examination*

Part Measured	Right Side, Inches	Left Side, Inches	Difference
Head, circumference	11	9¾	1¼
Width of orbit	2½	2⅜	⅛
Width across the shoulders	7	6⅝	⅜
Arm, length from the acromion process to the olecranon	12½	12¼	¼
Forearm, from the olecranon to the styloid process of the ulna	8¾	8½	¼
Arm, girth at shoulders	16	15¾	¼
Forearm, girth	10½	10⅛	⅜
Wrist, girth	7½	7¼	¼
Middle finger, length	3⅝	3½	⅛
Middle finger, girth	2½	2⅜	⅛
Chest, breadth	5	4	1
Abdomen circumference	14¾	12¼	2½
Abdomen from umbilicus to anterosuperior spine	5¼	4¼	1
Pelvis, circumference	16¾	13⅞	2⅞
Pelvis, external anterosuperior to internal	34	31⅞	2⅛
Thigh, girth	19½	17¼	2¼
Leg, girth at calf	12¼	11¼	1
Knee, girth	12⅞	12	⅞
Foot, girth	8½	8⅜	⅛
Leg, length	16⅝	15⅝	1
Length of big toe to heel	9⅜	9	⅜

CASE 2.—B. L., chronological age 12, mental age 6, intelligence quotient, 42; the second of three children; a full term, normal baby, weighing 7½ pounds (3.4 kg.); was breast fed for fourteen months. She first walked at the age of 18 months; she talked at the age of 2 years; her first tooth appeared at the age of 8 months; she had no diseases of childhood except chicken-pox and measles. There was no history of convulsions, fits, fainting spells, accidents or injuries of the head. The child began school when 6 years old, attended regularly, but was unable to get beyond the second grade; she was finally placed in an ungraded class, which she now attends. She forgot easily and was inclined to be lazy, but showed particular aptitude for the piano and had learned to play by ear anything she had heard once. She played harmoniously with children younger than herself. At times she was stubborn, quick tempered and quarrelsome; at other times, lovable and affectionate. There were no other disorders of conduct; she always told the truth, even though punished for doing something wrong. She slept well but talked in her sleep. She was very clean about her person, liked good clothes and was tidy.

TABLE 2.—*Measurements of First Patient at Last Examination*

Part Measured	Right Side, Inches	Left Side, Inches	Difference
Head, circumference	11	9¾	1¼
Width of orbit	2½	2⅜	⅛
Width across the shoulders	7½	7	½
Arm, length from the acromion process to the olecranon	12½	12¼	¼
Forearm, from the olecranon to the styloid process of the ulna	8¼	8½	¼
Arm, girth at shoulders	16¼	15¼	1
Forearm, girth	10⅝	10⅛	½
Wrist, girth	7¼	7½	¼
Middle finger, length	3¼	3½	¼
Middle finger, girth	2½	2⅜	⅛
Chest, breadth	5½	4	1½
Abdomen circumference	15½	12	3½
Abdomen from umbilicus to anterosuperior spine	5½	4¼	1¼
Pelvis, circumference	17¼	13¾	3½
Pelvis, external anterosuperior to internal	34	31⅞	2⅛
Thigh, girth	19¾	17½	2¼
Leg, girth at calf	12¾	11⅝	1⅛
Knee, girth	13	12	1
Foot, girth	8¼	8⅛	⅛
Leg, length	16¾	15⅝	1⅛
Length of big toe to heel	9⅜	9	⅜

TABLE 3.—*Measurements of Second Patient*

Part Measured	Right Side, Inches	Left Side, Inches	Difference
Head, circumference	10⅛	10	⅛
Width of orbit	2	1⅞	⅛
Width across the shoulders	6	5⅝	⅜
Arm, length from the acromion process to the olecranon	12⅛	12	⅛
Forearm, from the olecranon to the styloid process of the ulna	8	7⅞	⅛
Arm, girth at shoulders	15	14	1
Forearm, girth	6¼	6⅜	⅜
Wrist, girth	5⅝	5⅜	¼
Middle finger, length	3⅛	3⅛	¼
Middle finger, girth	2½	2¼	¼
Chest,, breadth	4½	3⅜	1⅛
Abdomen, circumference	13¾	10¾	3
Abdomen from umbilicus to anterosuperier spine	6	4⅞	1⅛
Pelvis, circumference	15	13	2
Pelvis, external anterosuperior to internal	30⅛	28	2⅛
Thigh, girth	14½	13½	1
Leg, girth at calf	12⅛	11⅛	1
Knee, girth	12½	11	1½
Foot, girth	7¼	7⅛	⅛
Leg, length	16	15¼	¾
Length of big toe to heel	8⅛	8	⅛

Physically she was well developed and well nourished, with overdevelopment of the entire right side extending from head to foot. The data presented in Table 3 disclose the condition.

She had an external strabismus with a high degree of myopia and chronic nasal catarrh with deviation of the septum. The canine and incisor teeth were in a double row, and there was a high arched palate. There were many pigmented nevi over the right leg and left hand.

The laboratory findings, including the blood and spinal fluid Wassermann tests, were normal.

Roentgenograms of the entire skeleton showed that the enlargement also involved the bones. The sella turcica was normal.

SUMMARY

Anatomic development anomalies are often associated with mental deficiency.[1] Of the forty-four cases reported in the literature up to date,[2] (*a*) six patients were adults, (*b*) thirty had hemihypertrophy on the right side, (*c*) fourteen had it on the left side, (*d*) twenty were males, (*e*) twenty-four were females, and (*f*) in five the condition was definitely associated with mental deficiency. Disturbance in the embryonic period seems to be the most reasonable etiology. Heredity seems to play an unimportant rôle.[3]

Cutaneous pigmentation is often present in hemihypertrophy.[4]

No treatment is necessary for the hypertrophy.

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Abstracts from Current Literature

CLINICAL AND EXPERIMENTAL STUDIES OF THERAPEUTIC INOCULATIONS OF MALARIA. BERNHARD DATTNER and OTTO KAUDERS, Jahrb. f. Psychiat. u. Neurol. **43**:1, 1925.

This investigation was conducted at the Vienna Psychiatric Clinic. Treatment with malaria was employed not only in the metasyphilitic diseases of the central nervous system, but also in multiple sclerosis and postencephalitic parkinsonism. It was also employed, though without results, in several cases of hebephrenia and especially in katatonic dementia. The successful results of malarial treatment in paresis, particularly as regards the four reactions, also led to its employment in cases of secondary syphilitis with positive spinal fluids as well as in cases of primary syphilis. Of the two tertian strains of malarial parasites employed at the Clinic, one was transmitted from man to man sixty-two times, and one seventy-seven times. Seventy-three direct transmisions through various hosts were made without the slightest change in the biologic manifestations, toxicity, infectiousness or morphology of the organism. Some of the strains employed at this clinic were also transported and successfully employed in neighboring states and foreign countries, the farthest being Denmark. Five hundred patients in all have thus far been treated.

Indications: Generally speaking, any person who, except for disease of the central nervous system, is free from primary acute or chronic disease or functional disturbance of any organ is adaptable to malarial therapy. Markedly debilitated and poorly nourished persons are not favorable subjects; not so much on account of the danger from complications as from their inability to withstand the necessary number of febrile attacks, so that treatment must be stopped before the desired results are obtained. Other contraindications are: febrile diseases, and severe affections of the heart, liver, kidneys, and spleen. Slight and compensated valvular lesions, such as mitral and aortic insufficiency, are not to be regarded as absolute contraindications; in these cases the danger of acute cardiac insufficiency and sudden collapse is not great. Myocardial degeneration with severe symptoms of insufficiency is a definite contraindication. Arrested tuberculous lesions in the lungs and other organs are not contraindications. Unusually obese persons must be watched especially during the febrile attacks, because in the authors' experience these patients collapsed entirely during the first attack of fever, from marked loss of strength, diarrhea and vomiting. In spite of all the contraindications mentioned, however, there is no great necessity to adhere rigidly to them, because in the event of the appearance of alarming complications during the febrile attack the latter can be promptly arrested by the administration of quinin. The youngest patient treated in the Clinic was 9 years old and the oldest 70.

Methods of Inoculation: The authors recognize four methods: (*a*) the usual combination of subcutaneous injection and scarification, (*b*) intravenous inoculation, (*c*) intracutaneous inoculation and (*d*) inoculation by scarification.

In employing the first method it is immaterial whether the plasmodium containing blood is taken during the onset, acme or decline of the fever or during an afebrile stage, provided the donor has had at least one typical attack of fever. In one case a positive result was obtained with blood taken from a

donor thirty-one hours postinfectionem. The method is carried out as follows: Under aseptic precautions from 2 to 4 c.c. of blood is withdrawn from the donor's cubital vein and immediately injected into the donee underneath the skin over the back; the needle is inserted deep into the subcutaneous connective tissue, and during the injection is directed in various directions in order to produce several small lesions in the vessels in order to insure the entrance of the parasites into the circulation. The second step consists in scarification of the skin; this is performed with a specially constructed lancet or with the injection needle, with which several short scratches are made in the skin of the back both longitudinally and transversely; this process necessitates an actual traumatization of the superficial layers of the skin; immediately after this traumatization a few drops of blood allowed to remain in the syringe after the injection are squirted into the scarified area; the blood is then allowed to dry, and the scarified area is covered with adhesive plaster. Local reactions following this method of inoculation were never observed. A certain amount of skill is essential in the technic in order to conclude the injection before the blood has coagulated, although unusual speed is hardly necessary, because injections were made successfully into two or three patients with the same syringe, the needle being changed in each case before the blood began to coagulate. No parallelism was observed between the time of onset of the reaction following inoculation and the quantity of blood injected. A positive result may be obtained with considerable smaller amounts of blood than from 2 to 4 c.c., without actually prolonging the period of incubation.

The second method finds its practical application in cases in which for some reason or other a shorter period of incubation — even only a few days — is necessary. From 0.1 to 10 c.c. (average 1 c.c.) of blood are directly injected from the cubital vein of the donor into that of the donee; with proper technic, defibrinization or the addition of solutions inhibiting coagulation is unnecessary.

For the intracutaneous method of inoculation, very small quantities of blood are necessary, from 0.1 to 0.5 c.c. being sufficient; the needle is introduced into the skin in a direction parallel to it, and the blood is allowed to run into the skin in quantities of about 0.1 c.c., so as to raise several small wheals. This method is indicated in cases in which a longer period of incubation is desirable.

Inoculation by scarification alone is not practical, except as it is used in connection with the first method, although Wagner-Jauregg himself has reported positive results following scarification, as far back as 1917.

Inoculation with "Conserved Blood": For a successful inoculation with "conserved blood" it is also immaterial whether the blood to be conserved is taken from the donor during an afebrile period, especially so when the question is one of "conserving" the blood only for a brief period. When, however, a longer period, twelve hours or more, is necessary, and particularly when the question of transporting the blood at body temperature is a vital one, the blood to be "conserved" must be withdrawn either during the acme of a malarial paroxysm or during defervescence, or, at least immediately after the cessation of the fever, i. e., at a period during which the sporozoits liberated during the fever are beginning to invade the erythrocytes, or when the small ring forms are just beginning to develop. Should there really be a question of "survival" of the plasmodia, it would seem that the young and resisting forms would naturally have the best chances for "survival."

Incubation, Prodromes, Febrile Onset: With the subcutaneous method, the average period of incubation would seem to be from six to eight days; a shorter period than this is extremely rare, but a longer period of incubation is common. Unusually long periods, from ten to twenty-one days, have been reported. These differences in duration of the period of incubation may possibly have been due to faulty technic, but are more likely to be attributed to factors to be sought in the inoculated persons themselves. What these factors may be is as yet unknown. No general rule can be given as to the duration of the period of incubation when the patients are inoculated intravenously, as compared to those inoculated intracutaneously; all that one can say at this time is that the period of incubation following the intravenous method is from two to three days shorter than that following the subcutaneous method; this, however, is not always true. A period of incubation of more than eight days is more rare in patients inoculated intravenously than in those inoculated subcutaneously. The intracutaneous method, as a rule, prolongs the incubation period up to ten days.

Most of the patients inoculated subcutaneously showed no reactions, as far as a rise in temperature was concerned; a small number, however, immediately after the injection, had a transitory rise, 38 C. (100.4 F.) being the highest temperature noted. Any rise in temperature immediately following the injections is regarded by the authors as a "protein" reaction. The period of incubation may further be characterized by prodromal rises of temperature up to 37.5 C. (only rarely over 38 C.); these are never typical; they can surely not be regarded as true febrile paroxysms, even though in some instances they are followed by more or less sweating; in some cases sweating has been observed during the period of incubation even without a preceding rise in temperature. Generally speaking, a rapidly increasing number of these febrile attacks with sweating and a gradual rise in the temperature curve with each successive attack of fever justifies the prediction that the typical tertian fever is about to appear. One is almost justified in regarding these prodromal fevers as "malarial equivalents." Subjectively most of the patients complain, at this time, of general exhaustion, prostration, anorexia and various sensory disturbances in the lower limbs, such as peculiar dull pains in the bones, especially the tibiae; these pains may occur even in nontabetic patients in "lightning-like" paroxysms, and may be lancinating in character; occasionally there are complaints of severe cramplike pains in the calf muscles. During the period of incubation, the presence of plasmodia cannot always be demonstrated in the smear, although occasionally during the fever two or three plasmodia may be found in a well prepared smear.

In a small percentage of cases (a fraction of 1 per cent.) the results of inoculation may be negative, although even these patients may have a transitory rise in temperature with sweating, but this never assumes the character of a typical fever. A repetition of inoculations in these cases was followed by no better results. This would appear to indicate a natural immunity against malaria.

The authors do not consider a febrile attack as a malarial paroxysm unless it is preceded by a chill and followed by a rise in temperature of at least 39 C. (102.2 F.) and sweating. Inoculation malaria is as a rule more "benign" than "natural" malaria; this is noted, for instance, in the shorter period of incubation (two and one-half days), which is never observed in "natural" malaria; clinically inoculation malaria runs a milder course; its complications

are always transitory and reparable. The character of the fever is different; the typical textbook curve of a tertian paroxysm is almost never observed in inoculation malaria; there are great variations in the course of the fever, such as an attack of tertian fever followed by one of the quotidian type, or vice versa; there are always variations in the duration of the fever; these variations find their explanation in the morphologic variations in the maturation of the protozoa, as observed in the smear. The possibility of personal dispositional factors to account for these variations must be borne in mind. As a general rule, blood smears taken during the febrile stage of inoculation malaria show a smaller number of plasmodia than in "natural" malaria; the Schüffner type of stippled erythrocyte found in "natural" tertian malaria is an inconstant finding in the smears in inoculated malaria; it may be found only during the first febrile attack or only during the last, or it may be absent.

As a rule two types of fever are observed. The first is a rapidly increasing rise in temperature which, after reaching a certain height, remains stationary for a few hours, and the second is a so-called "abortive" type of fever, in which a temperature of over 38 C. is reached, and which lasts only from three to four hours. The average duration of a febrile paroxysm (counting from the first rise until the temperature has become normal again) is from seven to eight hours. Temperatures over 39 C. usually last from three to four hours, although temperatures as high as 40 C. (104 F.) may occasionally last several hours; the highest temperature noted by the authors was 41.7 C. axillary, (107 F.). Fevers lasting more than eight hours are not rare. Occasionally paroxysms with lower temperatures would be interpolated between those with higher temperatures. The hour of onset of the successive paroxysm is indefinite; it can never be determined with the almost mathematical precision that it can be determined in "natural" malaria. The chill assumes a certain definite characteristic; it begins suddenly and is particularly severe when the initial incubation temperature is very high. It may last from one half to one hour. Some patients may have a less severe chill and one of shorter duration at the acme of the fever. In cases in which there occurs a gradual rise of temperature, the initial chill may be absent, or it may appear for the first time at the acme of the fever; in such cases the chill lasts only about ten minutes. As the fever declines, the patient begins to sweat profusely; the sweating also varies in intensity. The authors' patients in whom the sweating was slight or absent, showed evidences of collapse.

In some cases the febrile paroxysms would cease spontaneously, i. e., without the administration of quinin, but they could be brought on again by the administration of such provocative agents as protein preparations, a nonspecific milk preparation, typhoid vaccine and sodium nucleinate. As a rule, the fevers induced by these agents would again assume the typical type, but they would be much milder, and could hardly be regarded as classical attacks of malaria. The majority of patients were allowed to have eight typical paroxysms. Vigorous young persons who could tolerate the paroxysms without complications were allowed to have from ten to twelve attacks.

Some of the complications observed during the febrile attacks were acute gastro-enteritis or gastroduodenitis; some patients in the final attacks would develop a subicteric staining of the skin and mucous membranes — evidences of a slight passive congestion of the biliary tracts, or perhaps of a transitory injury to the hepatic parenchyma. Clay-colored stools and other pathologic manifestations of liver and gallbladder disease were not observed; urobilin was found to be increased in the urine. All these manifestations, however,

disappeared within a week after the general improvement following the postfebrile period was well advanced. Enlargement of the spleen, as far as could be determined by palpation and percussion, was slight; this was also corroborated in the cases which came to necropsy.

During the febrile period special attention must be given to the cardiac activity. Every one of the authors' patients was given ten drops of the tincture of strophanthus at the height of the paroxysm; more delicate patients were given two doses of this drug, and the administration of stronger cardiac stimulants was hardly ever indicated. Patients with well compensated cardiac lesions and older persons with evidences of beginning myocardial degeneration were given digitalis, both before and during the febrile paroxysms. The appearance of collapse, increasing debility, pallor, irregular heart action and paroxysmal tachycardia alternating with bradycardia and cardiac irregularity were indications for the immediate cessation of the treatment; in these cases oleum camphoratae combined with caffein was administered, to prevent and to treat the imminent colapse. Less than 1 per cent. of the authors' cases succumbed to cardiac collapse. Antipyretics, usually in great demand by most patients in order to overcome the disagreeable effects of the fever, are not only contraindicated, but are actually harmful, because they tend to decrease the fever and thus defeat the object of the treatment.

There is no unanimity of opinion as to the blood picture in cases treated with malaria. The usual leukopenia with an increase in neutrophils, as it occurs in "natural malaria," is also reported by most observers in inoculation malaria, and has also been found by Dattner and Kauders; these authors also find that as the leukopenia is increasing, there is a corresponding decrease in the number of red blood cells, though not perhaps as marked as in "natural" malaria.

The after-treatment consists in the administration of 0.5 gm. of quinin sulphate or hydrochlorid, twice a day, for three successive days, followed by 0.5 gm. once a day for four successive days — 5 gm. in all. This amount of quinin, given in this manner, was found sufficient to produce the desired results. The further administration of quinin will depend on the presence or absence of the malarial plasmodia in the blood. The patient's recovery, both objectively and subjectively, as far as the malaria is concerned, is most striking. Simultaneously with the administration of quinin, all patients, including those suffering from nonsyphilitic diseases, are subjected to arsphenamin therapy. Neo-arsphenamin is injected intravenously in weekly intervals, 0.3 gm. the first week, 0.45 gm. the next week and 0.6 gm. during the next four weeks. Debilitated persons are preferably given six weekly doses of 0.45 gm. each. In nonsyphilitic cases, (multiple sclerosis, parkinsonism) from two to three injections of arsphenamin are sufficient. The authors state that the possible beneficial effects of arsphenamin therapy on paresis treated by malaria is still a debatable question, the discussion of which is not within the scope of this paper. The administration of arsphenamin in patients inoculated with malaria is merely employed as an adjunct to the quinin therapy of the malaria.

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MIND BLINDNESS. NIESSEL VON MAYENDORF, Klin. Wchnschr. **4**:451 (March) 1925.

In this article Mayendorf discusses the optic agnosia of Munk under two headings: 1. What is meant by mind blindness in the psychologic sense? 2. What is its fundamental mechanism?

With the present status of our knowledge, these questions may be satisfactorily answered. First of all, the symptom must be sharply defined. The examiner must be certain that an optic disturbance is not causing the agnosia. Such cases as defects in form or color perception are not taken up in this investigation. Secondly, the patient with mind blindness must be perfectly normal mentally.

Agnosia may be macular (central), retinal (peripheral) or a combination of the two. Whether the clinical types represent these forms entirely or in part must be learned by experience.

In investigating mind blindness an endeavor must be made to determine whether there is a defect in the content of consciousness or the function of consciousness. Also it must be clearly determined whether the normally perceived objects are not recognized because the optic memory picture cannot be stimulated by the periphery, or because the picture has been lost. The optic agnosia may be the result of a thought disturbance which incapacitates the individual to carry out the intellectual identification process largely because of inability to concentrate, and because of lack of power to hold to an impression long enough to compare it with the corresponding trend of thought.

Consciousness which is a faculty of the cerebrum may be lost in a psychic sense only when necessary material fails. This is a primary loss; the second is the loss of identification ability. For this reason mind blindness must be looked on as a memory disturbance.

This knowledge directs the answer to the second question in that we now ask: Which are the brain centers that serve optic comprehension and optic memory, and which are the association tracts between these centers?

Up to the present time there has never been a case of isolated disease of the occipital cortex convexity observed with symptoms of mind blindness; the optic radiation of Gratiolet has always been affected. Numerous cases of mind blindness have been observed in which the affection was caused by disease of the left calcarine region with the occipital convexity intact. Double-sided affections of the calcarine cortex are accompanied by typical memory disturbances, and the cortical macula is likewise pathologically affected. This may result in the interesting picture of astereognosis of the contralateral hand, and warrants the hypothesis of the separation of the cortical areas of perception and memory. The appearance of mind blindness does not prove that the cortical center which stimulates memory has been destroyed, but it might well be assumed that long association systems join the optic area of the calcarine cortex with other areas and that these are broken down in mind blindness.

There are characteristic clinical peculiarities in mind blindness, namely, its incompleteness, its changeableness and its capability of partial restitution. It was this that caused von Monakow to look on mind blindness as functional

For twenty years the author has been working on a new mechanism of mind blindness based on three facts: (1) the appearance of mind blindness following disease of the occipital lobe, the parietal lobe or the temporal lobe; (2) the appearance of mind blindness only in such cases of parieto-occipital cortex affections which are associated with the optic radiation of Gratiolet; (3) the appearance of mind blindness in diseases of the left hemispere with the corpus callosum intact.

Further the author establishes his theory on three other facts which seem to be recognized by the majority of neurologists and ophthalmologists: (1) on the bilateral representation of the macula lutea, contrary to the unilateral representation of the peripheral surfaces of the retina; (2) on the central

projection of the retina in the calcarine cortex followed by circumscribed localization of the site of keenest vision and that in the occipital pole; (3) on the existence of a macular bundle of the optic radiation which ascends from the outer geniculate body and by a large arch descends through the temporal lobe on a slant, connecting it with the cortex of the occipital pole.

From the foregoing the following is concluded: Mind blindness is the clinical symptom in the breaking down of the left macular bundle of the optic radiation, or of the left central projection of the macula in the cortex of the occipital pole. 1. Central scotoma is not a constant finding in mind blindness, as the optic impressions of both foveae are taken up in the right macula. 2. The perception picture may remain normal but be detached from the left hemisphere, and therefore there may exist an association disturbance even though association tracts are not disconnected by a focus of disease. 3. Mind blindness appears only partial as is the rule in aphasic symptoms, as the right macula endeavors to take over the function of the left side. The right macula gradually succeeds in making connections with the centers of the left hemisphere. 4. Therefore, mind blindness presents a picture brought about by organic disease, yet functional in the sense that the right hemisphere may produce partial restitution.

The author refers to a case reported by Wilbrand and Sänger, in which a limited softening of the basal surface of the left occipital lobe was found which extended into the occipital pole. The patient did not recognize any of the objects exhibited to him. This case the author considers as an experimentum crucis.

MOERSCH, Rochester, Minn.

A COMPARISON OF INTERNATIONAL STATISTICS OF GENERAL PARESIS. KOLB, Ztschr. f. d. ges. Neurol. u. Psychiat. **96**:74 (April) 1925.

Kolb gathered statistics concerning paresis from countries throughout the world, and summarized the conclusions which he drew as follows:

1. It is undeniable that in the last decades a much higher percentage of people have died of paresis than in former times.

2. The increase in paresis in some countries such as Mesopotamia, Arabia, South China, etc., has been accompanied by a decrease in other countries—Italy, United States, and the Netherlands. The general conception hitherto has been that paresis was extant in the former countries but that paretic persons either did not come to physicians, or paresis was not recognized by physicians when seen. Against this argument is the fact that in European countries, paresis was absent only a relatively short time ago, and that in France it has spread rapidly in two decades. The curves of incidence of paresis in Chile, Buenos Aires, Palermo, Messina and Syracuse show definitely that the percentage increase of paresis has been very great during the past few years. In Buenos Aires, for example, the number of deaths from paresis on the basis of each million of inhabitants increased from eighty-nine in 1898 to 218 in 1907. In Palermo it increased from seventy-two in 1903 to 294 in 1906.

3. Under certain conditions in a country which has been free from paresis or in which paresis has been infrequent, this disease may develop rapidly to great proportions.

4. The frequency of paresis, outside of its dependence on the frequency of syphilis, is dependent also on a second factor which the author designates as *Y*. The rapid increase of paresis in certain countries must be directly dependent

on this factor. Wherever this factor *Y* is lacking or ineffective, paresis is lacking or infrequent even when the morbidity of syphilis is very high.

5. "Explosive" increases in paresis which are observed under certain conditions can quickly recede (Buenos Aires, Southern Italy) even if the incidence of paresis seems to be greater than before the exacerbation.

6. Paresis curves of certain countries show a gradual increase (Germany Austria, Hungary and Spain); other countries show fluctuation in the curve (Italy and the United States); other countries show a gradual increase without strong fluctuations (Switzerland, Norway, Denmark and Sweden).

7. In other respects the paresis curves of various countries show important differences with respect to the height and incidence of paresis. The differences in the frequency of paresis are often greater than the differences in the frequency of syphilis—here the factor *Y* is important. The curves of Sweden, Norway and Ireland are particularly low, while those of Italy, Budapest and Japan are high. The differences in the course of paresis over many years are more than can be explained by mere differences in the incidence of syphilis. Here too, *Y* is a factor.

8. The incidence of deaths from paresis among persons of various ages differs in different countries. Statistics of the United States and England show a striking incidence among the elder. This is true especially of tabes in the United States. The assumption that syphilis among the older classes is likely to lead to paresis finds strong support in the statistics of England and America. This high incidence among elderly people may be explained by the fact that (*a*) the average span of life is higher in the United States, (*b*) the average age of infection is higher in these places, (*c*) the average latency of paresis is higher, and (*d*) the disease runs a slower course.

9. Among males and females in the same country there can be found variations in the mortality from paresis. From 1908 to 1913 in the United States the average age of death from paresis for men was 50 years and 10 months, while for women it was 56 years and 10 months. For tabes it was 57 years for men and 56 years for women. Taking the average latency of paresis as fifteen years, the age of infection of men was 32 years, while that of women was 38 years.

10. Differences in the development and spread of paresis occur among different races in the same country. The spread of western culture is a factor here. In the United States paresis among the blacks was rare, despite the high incidence of syphilis, until a new generation had grown up in the country.

11. Women develop paresis later than men.

12. In countries in which the parasyphilitic diseases are new, tabes occurs earlier than paresis, owing to the shorter latency.

13. Deaths from tabes and paresis in all countries show that the number of deaths from the latter are from two to three times as great as from the former.

14. The period between infection and death is longer in tabes than in paresis.

15. When the curve of paresis is at a maximum in a country, the curve of tabes is at a minimum, and vice versa.

16. Europeans may develop paresis after infection if they go to a country in which paresis is lacking or rare.

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17. The percentage of deaths from paresis is not constant.

18. Concerning the frequency of syphilis, one may state that: (*a*) war increases the incidence of syphilis; (*b*) in the civilized countries before the world war there was a decrease in the incidence of syphilis; (*c*) the frequency of syphilis among women is greater than one might assume from the incidence of paresis among females.

ALPERS, Philadelphia.

THE STATUS MARMORATUS OF THE STRIATUM. CÉCILE VOGT, J. f. Psychol. u. Neurol. **31**:256 (June) 1925.

In 1911 the Vogts under the name of "l'état marbré du striatum" described changes in the striate body consisting of a replacement of many of its ganglion cells by myelin fibers. The number of the latter is far greater in this disease than normally, even if one takes into account the comparatively small size of the striatum. L'état marbré is regarded by the author as a malformation due to a dystrophy of the nerve cells; it represents one form of a group of diseases of the striatal system characterized clinically by a regressive (?) rigidity, the absence of a true paralysis, with the presence of more or less hyperkinetic phenomena, especially mobile spasms and athetoid movements.

The original classification of diseases of the striatum was based by the author on the characteristic of phenotypes, which was especially sought in patho-architectural peculiarities. By doing this, Cécile Vogt hoped to be able to distinguish the various forms of l'état marbré and to clear up its histogenesis. She also expected that by studying the hereditary factors in the families in which the disease occurred, as well as its clinical manifestations during the early months of life, she would be in a better position to ascertain more precisely the genesis of the different forms of l'état marbré. The relative frequency of the cases and the great variety of clinical pictures presented by them did not justify the conclusion that there existed one uniform cause for all cases; the ensemble of all forms of l'état marbré could best be explained on the theory of what the author calls "general specific pathoklise," i. e., a special property of the striatum to react to different noxa with patho-architectural changes of the same nature. Moreover, as the first case examined anatomically was hereditary, and because several cases of the disease may be encountered in one and the same family, it was concluded that this general specific pathoklise was frequently a congenital pathoklise.

Scholz observed two sisters, one of whom developed normally up to 11 months of age, and the other up to 10 months. Following a high fever, one of them began to show manifestations of a striatal syndrome characterized by akinesia and an intelligence defect; the symptoms did not correspond to those observed in l'état marbré, and Scholz could find no explanation for them in the anatomopathologic picture. Following trauma the second sister developed a similar, though less marked syndrome; this child was also mentally retarded. Pathologic examination of the first case showed l'état marbré. Sections stained by the Holzer and Alzheimer methods showed a considerable increase in the neuroglia and in the blood vessels—a dense network of neuroglial fibers. Scholz attributes the état marbré in this case to a destruction of cells and nerve fibers in islets which showed later an increased number of myelinated fibers due to a regeneration of the latter; but in spite of this regeneration the number of regenerated axis cylinders never became normal. Scholz admits that the large number of myelinated fibers found in this condition is due to a

myelinization of the axis cylinders which normally remain naked. In spite of this admission, however, he denies the embryonal development of l'état marbré, and discards Vogt's designation of l'état marbré, prefering to call it a "partial infantile sclerosis of the striatum."

According to the author, the foregoing designation is wholly inappropriate for the following reasons: 1. There is no doubt that there exists an état marbré which is fully developed before birth. The author now has under observation three cases in which the symptoms of the disease were present at birth, and in which no special exogenous factors can be found. In the seven cases previously published by her, the striatum was so diminished in size that there could be no question but that the disease must have developed during fetal life. Scholz, therefore, is not justified in speaking of the process in general as an infantile process. 2. Scholz's designation does not convey the conception which is characteristic of l'état marbré, namely, the hypermyelinization. 3. The term sclerosis is too comprehensive, because there is no evidence that the condition always advances to glial scar formation. The increased number of neuroglial cells and blood vessels found by Scholz in his case is not characteristic for most of the cases reported; the same may be said of the neuroglial fibers. One cannot, therefore, speak of a sclerosis in all cases of l'état marbré. Even in cases in which the ganglion cells in the hypermyelinated areas have been secondarily destroyed, no one can object to the view that a great number of axis cylinders have resisted the destructive process, as is the case in the various forms of status fibrosus.

Finally, the author calls attention to the fact that in the normal striatum there are always some small areas of myelinated fibers which can be distinguished, even macroscopically, from the remaining parts of the striatum. The number and intensity of these areas varies under normal conditions, but there is always a great difference between these and the areas found in cases of l'état marbré. Among the normal cases she includes several that have been referred to her to determine whether they present early stages of the disease. She is quite sure that should these cases in the future prove to be transitional types, their number will be insignificant as compared to the number of well developed cases that one encounters. This would seem to indicate that most cases of l'état marbré represent special genotypes. It would also seem that even though there may exist a purely acquired form, most cases are definitely an expression of a "genetic pathoklise" and must therefore be included among the malformations of the nervous system.

The article is in French.

KESCHNER, New York.

EXPERIMENTAL STUDIES ON THE PASSAGE OF ARSENIC INTO THE CENTRAL NERVOUS SYSTEM. HANS HOFF, Jahrb. f. Psychiat. u. Neurol. **42**:201, 1923.

Ehrlich did not include tabes or paresis in his list of indications for the use of arsphenamin, but neurosyphilis was soon treated by antisyphilitic measures, and some authors, such as Swift and Ellis, and Gennerich, advocated the use of arsphenamin in such a way as to bring it in close contact with nervous tissue. Wegefort and Marie injected it into the cisterna magna and ventricles; Knauer and Meggendorfer, into the carotids. Dercum, by spinal drainage after intravenous injections of arsphenamin, attempted to bring more arsenic in contact with the nervous system, and since this procedure Wagner-Jauregg has used malaria in the treatment of metasyphilitic conditions.

No attempt has been made to determine the efficacy of these treatments by animal experimentation, so that the author has set for himself the task of determining under what conditions arsenic reaches the nervous system following arsphenamin therapy, and what method of treatment is most efficient in bringing the greatest amount of arsenic into contact with the brain and spinal cord. For this purpose, he uses dogs that have received 0.15 gm. of neo-arsphenamin and that are killed by exsanguination. The experiments were carried out on twenty-five animals, and quantitative arsenic determinations were made on all the organs.

Intravenous injections of arsphenamin were given to a few animals. One animal killed after thirty minutes showed the greatest amount of arsenic in the intestines and liver; the meninges showed a relatively large amount, while the central nervous system and spinal fluid were arsenic-free. Another animal killed after two hours showed less arsenic in the meninges, while the liver contained the most arsenic and the kidneys some. After eight hours, the meninges showed only a trace of arsenic; the blood and other organs were arsenic-free. After twenty-four hours, the liver and intestines contained traces of arsenic, while the meninges contained none. In all these experiments the brain and spinal fluid showed no arsenic. Dilatation of the cerebral vessels by means of nitrates with the idea of causing a greater penetration of arsenic, gave negative results. One patient with a brain abscess showed arsenic in the walls of the abscess, but nowhere else in the brain. From his intravenous experiments the author concludes that arsphenamin is not neurotropic, that on intravenous injection it reaches the nervous system only under the rarest conditions, but that it reaches the other organs in abundance.

Intra-arterial (carotid) injections of arsphenamin gave results which were negative so far as the appearance of arsenic in the central nervous system and spinal fluid was concerned.

Intraspinal injections were carried out on dogs by the removal of 5 c.c. of spinal fluid to which arsphenamin was added, and the mixture reinjected. In this way from 0.0003 to 0.00075 gm. of neo-arsphenamin was injected. One-half hour after this injection arsenic was found in goodly amounts in the meninges, while traces were present in the liver and blood, and very faint traces in the kidneys; the brain showed no evidences of arsphenamin. After twenty-four hours, the meninges also showed no arsenic, and the liver was arsenic-free. Incidentally, the author found evidence of severe meningeal reactions after intraspinal injections, as evidenced by increases in protein and cells.

Subdural injections of arsphenamin resulted in the recovery of arsenic only in the meninges at the base of the brain. The brain itself was free from arsenic.

Extensive experiments were carried out on dogs treated with intravenous injections of arsphenamin and lumbar drainage. Lumbar drainage was performed ten minutes after the intravenous injection, and arsenic could at this time be recovered from the central nervous system. Even after twenty-four hours arsenic could be recovered from the brain and cord. The arsenic was present in amounts equivalent to 0.0025 mg. arsenic for every gram of nervous tissue. The author concludes from animal experimentation that lumbar drainage after intravenous injection of arsphenamin is the only one of the present methods which brings arsenic into contact with the central nervous system.

ALPERS, Philadelphia.

THE REFLEXES OF THE COENESTHETIC SENSE, WITH OBSERVATIONS ON THE RUDIMENTARY REFLEXES IN MAN. JOHANN SUSMANN GALANT, Jahrb. f. Psychiat. u. Neurol. **43**:235, 1924.

The coenesthetic sense, considered in the light of a psychosomatic function of the organism as a whole, is one of the most delicate senses. The slightest and most delicate variation in the psychic or in the somatic components of the organism is immediately perceived by the coenesthetic sense, so that the latter may truly be characterized as a finely adjusted stabilizer of the psychosomatic equilibrium of the individual. Its activity is manifested by certain reflexes, and, as a matter of fact, its manifestations in the outside world cannot be anything but reflexes, because they have nothing to do with functions controllable by the will; they are psychosomatic processes, independent of volition, and reacting to stimuli which originally unconscious have never reached consciousness.

Coenesthetic reflexes are not mimicsomatic reflexes in von Bechterew's sense, although they may be of fundamental significance even as far as these reflexes are concerned. Von Bechterew designates as mimicsomatic reflexes almost all physiologic manifestations of sorrow and joy, disgust and pleasure, anger, anxiety, fear, surprise, repentance, etc. It is self-evident that there may also occur among the coenesthetic reflexes physiologic manifestations similar to those noted among the mimicsomatic reflexes. Coenesthetic reflexes are characterized by the fact that they are never manifested locally; they always find expression in a reaction that involves the entire organism; they are usually represented by a certain attitude assumed and maintained by the body for a considerable period, and they generally have a psychic background of no little significance to the organism.

In some instances a coenesthetic reflex may express itself in the form of an attitude by one who is "pulling himself together." An individual who is suddenly exposed to an explosion will react by "pulling himself together"—a reflex usually associated with a mimicsomatic expression of fear, surprise or even death agony; frequently the individual, stunned by the suddenness of this unusual experience, will maintain the attitude assumed for a considerable length of time. At times one has to deal with a coenesthetic reflex associated with the reflex of "listening to." The reflexes of "pulling one-self together" and of "listening to" are especially marked in animals; in man they are atavistic and more or less rudimentary in nature. Although these reflexes, especially in animals, are manifested by a certain attitude which is assumed by the entire body, nevertheless it will be found that there is a particular segment in which the manifestation is most evident; this is the so-called "concentration point of the reflex." A dog, for example, will hold his entire body rigid during the reflex of "listening to," but especially so his ears—he will "prick up his ears"; the ear then is, in this instance, the concentration point of this particular reflex. These reflexes are of special significance in beasts of prey. Birds, mammals and reptiles assume involuntarily a peculiar attitude as if they were throwing themselves on their victims.

Another group of reflexes belonging to this category are the so-called "feeling reflexes"; they are either defense or surprise reflexes. In man the surprise reflex is a rudimentary reflex. Man usually learns to assume various attitudes defensive in nature, during military exercise, boxing, fencing, etc.; sooner or later these attitudes become automatic. Other "feeling reflexes" observed in man are lightning-like twitchings of groups of muscles, blinking of the eyes, marked nystagmus and other similar nervous manifestations. The

author also mentions many other reflexes of "feeling," such as the well-known reflexes of hunger, thirst, sexual excitement, etc., whose point of origin is in the internal organs; these, however, he promises to discuss in detail in a future communication.

KESCHNER, New York.

THE MYOCLONIC FORM OF ENCEPHALITIS. WERNER REIMOLD, Ztschr. f. d. ges. Neurol. u. Psychiat. **95**:21 (March) 1925.

The question of myoclonus has been an open one since Friedreich's contribution on paramyoclonus multiplex in 1881, and since Unverricht reported on the familial type of myoclonus. Encephalitis has of late years again directed attention to this problem. Mann reported eighty cases of encephalitis in the epidemic of 1922-1923, in 38 per cent. of which there was myoclonus either in pure form or in combination with paralysis agitans, chorea or athetosis. The occurrence of myoclonus was particularly frequent in the epidemic, while paralysis agitans was not common. A review of the findings since the first publication of Economo in Vienna and of the French in 1915-1916 shows that lethargy was the outstanding feature of the first epidemic, while in the succeeding epidemics postencephalitic disturbances became more prevalent, especially parkinsonism. Myoclonus was at first infrequent, but gradually increased in incidence until in 1919-1920 it was frequent. Grünewald reports an incidence of myoclonus in 32 per cent. of encephalitic patients up to 1922-1923. Wechsler found that a combination of psychic symptoms and myoclonic twitchings characterized the last New York epidemic. Crispel found myoclonic twitchings in twenty-three of forty-two patients from January to March, 1923. Hunt found "hyperkinetic symptoms" frequent during the same period. Smith, in an epidemic in the Philippines from November, 1922, to early in 1923, observed a predominance of hyperkinetic, myoclonic symptoms.

The Breslau epidemic, which the author reports, reached its peak simultaneously with that of the grip, which was toxic in nature. The disease came on either slowly with uncertain feelings of malaise, with headaches over a long period, later on vertigo, vomiting and fever, with the final onset of clonus after days or weeks; or its onset was acute, with severe pains in the muscles, which after a short time showed twitchings. In some cases the onset was subacute, with psychic disturbances, delirium, confusion, high fever and, sooner or later, myoclomus.

The muscles most frequently involved in myoclonus are the adductors of the upper arm (pectoralis major), the flexors of the forearm (biceps, brachialis and brachioradialis), together with the superficial and deep flexors of the wrist, and the interossei. The combined contraction of these muscles produces a characteristic rhythmic clonus with powerful adduction of the upper arm and flexion and pronation of the forearm. Through simultaneous contraction of the interossei a picture is produced almost similar to that seen in parkinsonism. Synchronously one often finds clonus of the respiratory muscles, which Stern has described under the term polypnea. The diaphragm, intercostals and subsidiary muscles are involved, and contract from fifty to seventy times a minute independent of and uninfluenced by volition. Elevators of the shoulder girdle may be likewise affected, e. g., the trapezius, levator scapulae, rhomboids, etc., contraction of which produces elevation of the shoulder. The muscles of the lower extremity are less commonly involved. Of the muscles of mastication, the masseters and temporals are most frequently affected, at

times also the pterygoids. In the neck, the sternomastoids are most often affected. Clonus of the abdominal muscles was infrequently seen. Particular attention is called to the combination of myoclonus with fibrillary twitchings (myokymia).

ALPERS, Philadelphia.

STRUCTURE AND LOCALIZATION OF GLIOMAS WITH SPECIAL CONSIDERATION OF THEIR DEVELOPMENT. DONALD J. MACPHERSON, Arb. a. d. neurol. Inst. Wien. Univers. **27**:123 (May) 1925.

A survey of the literature discloses three different theories as to the genesis of glioma. One theory is that the ependyma derived from the neural tube proliferates, or is capable of a proliferating process that may go on to the formation of glial tissue. The second theory, diametrically opposite to that above, assumes that the ependyma becomes anaplastic from the glia. The third theory regards the ependymal islets in glioma as a secondary mechanical or inflammatory process. Of great significance in all these theories is the occurrence of peculiar small proliferations near the cerebral ventricles. Judging from the opinions given by various observers, it is impossible to state definitely whether these little proliferations are really large-sized ependymal granulations, or whether they are not genuine new growths in the vicinity of the ventricles. Another important factor in this connection is whether these small neoplasms are not to be regarded as metastases or whether what is usually described as metastases in glioma is not really an expression of general maldevelopment.

From his own studies MacPherson is led to believe that the occurrence of these very small gliomas near the ventricles is an expression of an ontogenetic disturbance, showing that what is generally designated as glioma is not a new, localized formation of neoplastic tissue, but an expression of anomalous development in various parts of the brain and that these differ mainly on account of the varied intensity of the anomalous development. This conception finds substantiation in the fact that such small derivatives of the neural tube and its vicinity may be found isolated in the cortex, far away from the ventricle, without giving rise to true tumor formation. This would also explain the occasional multiplicity of these tumors and the transition to cases in which there is a diffuse gliomatosis. The fact that such nodules have also been found in neurinoma (Bielschowsky and Henneberg, Nishikawa, MacPherson) lends additional proof to the theory of dysontogenesis.

The origin of glioma is determined by two different factors. One of these determines its structure, i. e., the combination of ependyma cells and spongioblasts; the structure of the tumor then will depend on which of these elements predominates. The second factor is the stage of development of the tumor cell, be it ependymal or spongioblastic in nature; this determines the cellular structure as far as the size and maturity of the cells is concerned; as a result of this factor we see tumors with a predominance of small glial cells, or giant cells or plasma cells or tumors in which there is an extensive production of fibers at the expense of the cells. This factor justifies the designation of these tumors as gliomas in spite of the entirely different cellular structure presented by them. The only definite criterion as to whether a tumor is a glioma, is whether or not it bears a genetic relationship to a maldevelopmental defect of the neural tube, and if this cannot be determined from the tumor structure the evidence will have to be sought in the regions adjacent to the ventricles or in the brain substance itself. With this conception in mind, glioma is to be regarded as a tumor in which maldevelopment gave rise to neoplastic formation.

The concluding part of the paper is devoted to a discussion of the relationship of neurinoma to glioma. MacPherson describes a case in which he found a neurinoma in the hypothalamic region, which on microscopic examination showed glial cells and a cyst within the tumor itself. His discussion of this relationship is practically the same as that given in the body of the paper. The paper is well illustrated.

KESCHNER, New York.

DYPSOMANIA AND PSYCHANALYSIS. A. KIELHOLZ, Schweiz. Arch. f. Neurol. u. Psychiat. **16**:27-35, 1925.

The psychanalytic literature contains little concerning alcoholism. Freud himself does not touch on this subject directly. As is well known, he regards the sucking of infants as a sexual activity based on a constitutionally accentuated erogenous oral zone, and that a continuation of this tendency would result in oral masturbation, and in men would furnish a strong motive for drinking and smoking. Ferenczi, basing his opinion on the analysis of a case of alcoholic paranoia, stresses the importance of homosexuality in the genesis of jealousy in alcoholic persons. He regards alcohol as a disturber of sublimation. Bleuler questions the importance of alcohol itself as a mechanism in bringing about retrograde sublimation; he does not believe that the causal relationship between complexes and dypsomania has been proved.

A rather striking omission in the literature concerning this subject is the relationship between alcoholism and fraternal organizations. It is beyond question that the custom of drinking is both a product and a contributor to fraternal intercourse, and has always been so. It is this relationship which is responsible for the difficulty of combating the two. Julius Berger emphasized the association of this homosexual tendency with alcoholism. Freud remarked that homosexuality is more compatible with the understanding of mass psychology than a heterosexual libido would be. Kielholz does not want to be understood as inferring that homosexuality is present in every alcoholic person. He does, however, believe that it plays an important part in chronic alcoholism. The jealousy of alcoholic persons, which is so common, undoubtedly is easily explained on the basis of accentuated homosexual tendencies. In many alcoholic persons we must accept a constitutional prominence or fixation of narcissism. The prognosis in these cases is probably unsatisfactory. Many of the offspring of drunkards develop a hatred for the father because of his abusive behavior. This reaction against the father, however, is accompanied by a feeling of guilt, which brings about a depression and may in turn lead to comfort to be found in the cup that cheers. Kielholz believes that this is just as important in the causation of alcoholism as is heredity.

The hallucinations of delirium tremens are regarded as being peripheral in origin rather than the product of complexes; in those of febrile delirium, the latter is supposed to be the case. The writer cannot share this opinion entirely, for on analysis of patients who have suffered from delirium tremens he discovered a deep-seated connection between the early experiences and phantasies of these persons and the character of the hallucinations.

The study of exhibitionism has taught us how often this perversion is associated with alcoholism. The frequent occurrence of perversions in alcoholic persons has recently led Hans Sachs to classify psychopathic alcoholic patients as standing between the compulsion neuroses and the perversions.

In the treatment of alcoholism, Kielholz does not stress psychanalysis. It might be of value in cases complicated by outstanding neuroses. Pewnitsky

is the only writer who has advocated its use, although he stresses the importance of temperance associations. Narcissistic alcoholism has a poorer prognosis but this type of patient may be held best by providing him with a responsible position that would satisfy his ego.

WOLTMAN, Rochester, Minn.

THE SEROLOGY OF PARESIS TREATED BY MALARIA. LUDWIG HORN, Jahrb. f. Psychiat. u. Neurol. **43**:247, 1924.

This contribution deals with fifty-eight cases of paresis and twenty-one nonsyphilitic cases (multiple sclerosis, syringomyelia, encephalitic conditions) in which the patients were subjected to malarial treatment. The nonsyphilitic patients were inoculated with syphilis-free malarial blood. In these cases the blood Wassermann reaction was positive after a number of febrile attacks (as a rule, six such attacks were necessary, and in one case of dementia praecox, in which the Wassermann reaction was negative before the treatment, eight febrile attacks were necessary). Meinicke's flocculation reaction also became positive, but usually not until after the seventh attack of fever. The positive reactions persisted as long as the febrile attacks lasted; they disappeared after the first treatment with quinin. The positive Meinicke reaction usually became negative after the first half gram of quinin was given, whereas the Wassermann reaction would not become negative until after the second half gram. Of interest was also the fact that in the cases in which the administration of quinin, after at least eight or nine febrile attacks, was followed by a negative Wassermann test, which would remain negative for from nine to twelve days, it would again become positive after this period, in spite of the intravenous administration of neo-arsphenamin. The positive Wassermann reaction in the nonsyphilitic cases during the febrile period is believed most likely to be due to an enormous increase in plasmodial antigens. This would also explain the parallelism between the disappearance of the plasmodia and the sudden appearance of the negative Wassermann reaction. The second appearance of the positive Wassermann reaction is believed to be due to the formation of antibodies for the production of which a longer period is necessary, as well as to the fact that after the antibodies have once appeared they circulate in the blood for a much longer period. This naturally raises the question whether in these cases we are not dealing with a chronic form of malaria which gives rise to a positive Wassermann reaction. The absence of fever and of plasmodia in the blood smear would speak against such a hypothesis. The paretic patients after and during malarial treatment also showed more positive Wassermann and Meinicke reactions than before treatment was begun, i. e., when they had not been completely positive originally. The reactions after treatment did not run parallel with the clinical picture. Thus, cases were observed in which exquisite remissions occurred, but the Wassermann test remained strongly positive, although here too, as in the nonsyphilitic cases, the Meinicke reaction was somewhat weaker immediately after the completion of the treatment, and would become more positive or regain its original intensity only after a few days had elapsed after the completion of the cure. In paresis, genuine negative serology was not observed until from three to four months after completion of the treatment. It was at this time that the reactions began gradually to become weaker and in some instances became completely negative. The cases with such gradual improvement in the reactions were especially favorable from a prognostic point of view. As far as the albumin

and cell count of the spinal fluid is concerned, Horn finds that an increase in both of these appears to be an expression of a nonsyphilitic inflammation which has nothing to do with the acuteness of the syphilitic process.

KESCHNER, New York.

BRAIN TUMOR: NEURO-OTOLOGICAL EXAMINATION IN SEVEN CONSECUTIVE VERIFIED CASES IN THE POSTERIOR FOSSA IN CHILDREN. D. E. S. WISHART, Arch. Otolaryngol. **2**:1 (July) 1925.

The author gives seven full case reports of brain tumor in children. The final diagnosis was: (1) intracerebellar tumor on the right side with a cyst on the left, (2) glioma on the right side of the cerebellum with cystic degeneration, (3) papilloma of the choroid plexus of the fourth ventricle, (4) gliomatous cyst of the right cerebellar hemisphere, (5) glioma of the right side of the cerebellum, (6) gliosarcoma between the two halves of the cerebellum, (7) gliosarcoma of the pons and medulla. In the first case a study of the Bárány findings led to a correct diagnosis; in the third case, to the diagnosis of increased intracranial pressure chiefly in the posterior fossa; in the sixth case, to increase of intracranial pressure due to subtentorial lesion, and in the seventh case, to lesion in the posterior fossa. In the second, fourth and fifth cases, the Bárány findings led to the conclusion that the left side was chiefly affected, but necropsy showed tumor of the right side. Thus in seven cases the otologist gave the correct side once, the wrong side three times, and in three cases reported posterior fossa lesion without designating the side. In the latter cases the lesions were found to be: (1) in the fourth ventricle, (2) between the two halves of the cerebellum, (3) in the pons and medulla. The neurologist diagnosed the correct side twice, did not designate the side four times and in one case in which the tumor was found between the halves of the cerebellum noted that the right side was more involved than the left. In the three cases in which the otologist diagnosed the wrong side there was marked evidence of involvement of both vestibular tracts, but the vertical canal tracts on the left side showed less response to stimulation than those on the right side. This has led the author to the conclusion that "unilateral intracerebellar tumors will be found on the side opposite to the vertical canals with the function of which there is most interference."

The author's cases occurred in children aged 7 or under, and were very difficult to examine completely. There had been a decompression operation in Case 4 and the patient in Case 5 was examined six weeks prior to death. For these reasons it would appear unwise to make any general rule without further data.

HUNTER, Philadelphia.

PARESTHESIAS AND SOMATIC HALLUCINATIONS. JOHANN SUSMANN GALANT, Jahrb. f. Psychiat. u. Neurol. **43**:190, 1925.

Galant points out that it is as yet unknown whether somatic hallucinations, and for that matter hallucinations in general, are idiogenous or whether they originate in connection with abnormal processes in the organ in which they occur. He believes that many somatic hallucinations, if not all hallucinations, depend on paresthesias and perhaps other organic disturbances, but that they cannot detach themselves from the source of their origin, and therefore become idiopathic hallucinations. There is no doubt as to the occurrence of idiopathic hallucinations, even though the nonidiopathic variety would seem to be more common. The definition of a hallucination as a perception not based on a

corresponding environmental stimulus is subject to great limitations, because in many instances hallucinations are based on physiologic stimuli, even though the later are not always entirely adequate; but even in the absence of an adequate stimulus the faulty perception is an hallucination and not an illusion. The absence of a corresponding stimulus for a given false perception is not the most characteristic criterion that the latter is an hallucination. The proper criterion is the fact that a previous experience of certain perceptions, which, in the absence of a corresponding stimulus in the environment, is incapable of becoming an objective reality. For the development of an hallucination, a specially labile, vivid phantasy is an absolute necessity; the phantasy must be one which is peculiar to the autistic psychotic person who is so shut off from the rest of the world that he has lost all sense of reality. It is a realization phantasy which utilizes every slight abnormality in the sphere of general sensation and in the individual bodily organs in order to enable the individual to realize a sensational hallucinatory reality. It is also this mechanism which causes paresthesias to become the source of painful hallucinations. With the disappearance of the paresthesias, the hallucinations usually also vanish; in other cases the hallucinations last longer than the paresthesias which are at their basis, and then we have a more idiopathic if not idiogenous type of hallucination; the latter then persists independently of the paresthesia which provoked it, and which at this time has ceased to exist.

KESCHNER, New York.

MEDICO-LEGAL NOTES. Editorial, J. Ment. Sc. **71**:494-558 (July) 1925.

There is unusual interest in the evidence presented by the Medico-Psychological Association to the Royal Commission on Lunacy and Mental Disorder. The association states that the community in general is grateful for the protection afforded under the existing Lunacy Acts, but that these have failed to keep pace with medical progress. Its recommendations (fifty-eight) in general copy the best features of the United States practices. In discussing voluntary admissions, the statement is made that there seems to be no good reason why this mode of admission should be reserved for persons who cannot be certified as insane, as it conflicts with "the fundamental principle that treatment should be begun at the earliest possible moment." The tremendous prejudice against certification in Great Britain is partly explained by recommendations to drop the terms "lunacy, asylum, pauper" and to add licenses for the smaller sanitariums. One would expect intense objection to certification in Iowa, which now seems to mean that the person certified is listed as being unable to contract marriage.

In the opinions of witnesses selected by the Association, the treatment of new patients in such a way that they never see or know of the great mass of long resident patients is exceedingly well defended. The present teaching of psychiatry to the average medical student is regarded as inadequate to the point of absurdity—hardly better than it was thirty years ago. Dr. Menzies brings out the interesting point that public agitations for the improvement of conditions surrounding patients with mental diseases come once in every forty years, and that at these times of unrest laws should be moved forward a generation. Col. Lord reports that France is now considering its first voluntary mode of admission—"spontaneous internment" for the willing or the not unwilling, the latter class being very important. General hospitals in Holland are allowed to receive only two certified cases at a time.

The recommendations if adopted will do much to unify British and United States procedures, for there is a greater similarity in procedures in the larger states than appears on the surface.

BOND, Philadelphia.

THE CEREBROSPINAL FLUID IN GENERAL PARALYSIS TREATED WITH MALARIA. LUDWIG HORN, Jahrb. f. Psychiat. u. Neurol. **44**:83, 1925.

Pötzl, Fischer and Herrmann have shown that the hemolysin reaction in paresis became negative after treatment with malaria. Bearing this fact in mind, Horn investigated this phase of the problem of paresis in the large material at his disposal in the Wagner-Jauregg Clinic and in the Insane Asylum at Steinhof. The patients were subjected to the hemolysin test immediately after treatment, and the test was repeated several times later. As a result of his investigation he finds that the hemolysin reaction is a good index of the activity of the paralytic process. This is of great importance, because we know that the Wassermann and globulin reactions in paresis may remain positive for a considerable period after treatment with malaria, the positive results of these reactions being partly due to the direct effects of the malarial infection. The prognostic index of the hemolysin reaction, however, becomes less valuable, because Fischer, as well as this author, found that a positive hemolysin reaction that had become negative following malarial treatment, could, after a time, become positive again. A positive hemolysin reaction in cases in which there is a good remission would seem to speak in favor of a severe involvement of the meninges. A negative hemolysin reaction in definitely active processes may possibly be attributed to faulty technic, in which case the test is to be repeated several times; or else it may be due to the fact that there exist certain cases in which the blood is deficient in hemolysin. The many negative results obtained by the author in his group of demented paretic patients, he thinks, may possibly be an expression of a negative allergy. A negative hemolysin reaction in cases in which it had been positive prior to treatment went, in most cases, hand in hand with a decrease in the cell count and albumin content of the fluid. Some of the cases in which there was marked involvement of the vessels yielded a positive hemolysin reaction for a considerable period. The ease with which the test can be performed recommends it as a valuable guide in the determination of the progress of the paretic process, especially in conjunction with other fluid reactions and the clinical findings.

KESCHNER, New York.

DYNAMIC STUDIES ON THE CEREBROSPINAL FLUID IN THE DIFFERENTIAL DIAGNOSIS OF LATERAL SINUS THROMBOSIS. GEORGE L. TOBEY, JR., and JAMES B. AYER, Arch. Otolaryngol. **2**:50 (July) 1925.

"Compression of the internal jugular veins causes increased intracranial pressure with immediate rise in spinal fluid pressure." The absence of this rise indicates spinal arachnoid block or tumor of the cerebellar fossa. These facts are used in a new ingenious test for lateral sinus thrombosis. The patient is placed in the lateral position and the pressure of the spinal fluid noted. The jugular veins are alternately compressed and the effect on pressure noted. "In a typical case of lateral sinus thrombosis, there is a prompt and rapid rise of fluid pressure to twice or three times the initial reading when the jugular vein draining the normal sinus is compressed. This pressure rise is maximal, being equivalent to the pressure attained when both jugular veins

are compressed." "Pressure over the vein draining the thrombosed lateral sinus causes either no rise or, more commonly, a slow rise of only from 10 to 20 mm. in the manometer." Absence or rise due to low pressure or thick neck and abnormal rise due to improper technic may be seen in cases without thrombosis. There is a normal variation in pressure on compression between the two sides, but over 50 mm. is unusual and over 100 mm. is exceptional. Variations in size of the two lateral sinuses may exist, but are rare.

Spinal puncture should not be made if subtentorial abscess or tumor is suspected. The authors do not think there is any danger of starting a meningitis, if only small amounts of spinal fluid are withdrawn. They have made more than 3,000 spinal punctures and have had a series of twenty-two cases for diagnosis. Their success has been remarkable, as shown by a short list of case records, and by diagnoses such as the following: double mastoiditis with sinus symptoms, correct selection of the side involved; simple mastoid operation followed in three days by chills, positive blood culture and septic temperature, normal manometer readings, subsequent erysipelas; acute mastoiditis with septic chills, manometer readings normal, simple mastoidectomy, recovery; sinus case showing slight rise and slow fall of pressure on affected side, mural clot diagnosed and found.

This article is an important and new contribution and should be read in full.

HUNTER, Philadelphia.

THE NURSING REFLEX IN APRAXIA. STEFAN BETLHEIM, Jahrb. f. Psychiat. u. Neurol. **43**:226, 1924.

A woman, aged 62, with a left-sided hemiplegia, presented an exquisite motor aphasia with a high grade apraxia involving the mouth and a slight sympathetic apraxia in the right upper extremity. In spite of her age, there was considerable regression of the aphasia; three months after admission she showed in addition to agraphia and alexia only slight disturbances in speech —a slowing of spontaneous speech and an occasional difficulty in finding words and in naming objects. The hemiplegia, as well as the apraxia in the region of the mouth, was somewhat improved, but the sympathetic apraxia remained stationary. The aphasia in a right-sided lesion is explained by the author on the theory that the patient was probably left-handed (latent).

On admission it was found that owing to the apraxia the patient could not open her mouth when ordered to do so, although at first she snapped at objects (finger or keys) held in front of her. Then she would open her mouth without snapping it, and occasionally she would also protrude her tongue, and finally she would open her mouth completely, though slowly and feebly. According to Betlheim, this phenomenon represented a series of instinctive, complex and coordinated movements to an external stimulus, and is regarded by him as an "instinctive movement." This movement has previously been observed in the terminal stages of paresis and in dementia due to senile arteriosclerotic cerebral atrophy. Stransky designates it as a "nursing reflex" and regards it as a psychic reflex deeply buried since birth which comes to surface during involution, when all higher psychic levels have been destroyed. Most authors describe the presence of this reflex in more or less demented patients, and Betlheim lays special emphasis on the fact that his patient had no defect of intelligence; her attention could be attracted and maintained and she showed interest in her surroundings; she presented no evidences of asymbolia. He attributes the nursing reflex in this case to an injury to the praxia center for

the musculature of the mouth. This explanation, he believes, confirms the general rule laid down by Jackson and by Monakow that "with the destruction of a higher function there is a reappearance of a similar but more primitive function."

KESCHNER, New York.

FIBRO-CHONDROMA OF THE CAUDA EQUINA. F. L. A. GREAVES and F. G. LESCHER, Brit. M. J. **1**:592 (March 28) 1925.

These authors report a case of spinal cord tumor in a man, aged 42, who first complained of pain in the lumbar region four years prior to this time. After treatment for his general health he improved, but two years later gradually became worse. One year prior to admission to the hospital he noticed weakness in the right leg and some numbness about the perineum. Later in the same year he complained of inability to void urine. On examination he showed marked weakness in the right leg with slight muscular wasting and loss of muscular tone in this limb. There was pain on pressure over the right sciatic nerve, which was increased on flexing the thigh on the trunk and extending the leg on the thigh. There was definite impairment of sensation and loss of epicritic and protopathic sensation, with superficial pain and temperature sense which extended from a point just at the upper border of the right calf up the thigh in a narrow strip and over part of the right buttocks, with a saddle-shaped area which showed some loss over the left gluteal region. There was also loss of sensation over the perineum, scrotum and penis, with retention of deep pain and joint sensation. The mucous membrane of the urethra, lower rectum and anus were insensitive. The abdominal and cremasteric reflexes were active; the knee reflexes were distinctly diminished on the right. Both ankle reflexes were absent. Lumbar puncture gave a normal, clear spinal fluid without color. At operation the boney arches of the fourth and fifth lumbar and first sacral vertebrae were removed. On opening the dura a tumor described as leech-shaped was found beneath the dura, slightly attached to the inner surface of the dura by a tail. The patient improved gradually, except for mild retention of urine with overflow. Microscopically, the tumor is described as a chondroma with areas of necrosis and near the periphery some calcification. There was extensive hemorrhage into the fibrous tissue.

POTTER, Akron, Ohio.

THE PROBLEM OF CEREBRAL SYPHILIS AND GENERAL PARALYSIS AND THE PATHOGENIC SIGNIFICANCE OF THE ROUTE OF INVASION. H. SPATZ, Schweiz. Arch. f. Neurol. u. Psychiat. **16**:153-154, 1925.

Experiments have shown that there are two ways in which injurious substances may enter the brain: one source is from the spinal fluid and the other from the blood vessels. The encephalitic changes in syphilitic meningitis occur at the outset always and later chiefly in the superficial portions of the cortex and of the ventricular walls. In both spirochetal and dye produced meningitis the deeper portions of the brain escape for some time. The syphilitic endarteritis of Heubner ultimately brings about nutritional disturbances which may involve the deeper structures. With few exceptions, we must assume that in these cases the infection spreads to the brain from the cerebrospinal fluid.

A different distribution of the inflammation is seen in general paralysis. Here the gray masses, particularly the cortex and the related striatum, are involved equally. This picture cannot be explained by an introduction of the

virus from the spinal fluid. What one must assume is that the spread occurs through the blood vessels. The pathogenic difference between cerebral syphilis and paralysis does not depend on a difference of affinity of the toxin to mesodermal or ectodermal tissues, but on a difference in the portal of entry. We do not yet know what happens to the protective mechanism between the blood vessels and the brain substance in a patient who develops general paralysis, but must assume that a peculiarity of the spirochete or a congenital or acquired constitutional factor of the organism exists.

WOLTMAN, Rochester, Minn.

THE INFLUENCE OF THE CEREBELLUM AND CEREBRUM ON THE CENTERS FOR STATIC INNERVATION. W. J. BERNIS and E. A. SPIEGEL, Arb. a. d. Neurol. Inst. Wien. Univers. **27**:197 (May) 1925.

Among the rhombencephalic centers for static innervation, the vestibular nuclei play an important, though not exclusive rôle. Unilateral destruction of Deiter's nucleus and of the cell groups in the descending root of the vestibularis is followed by a definite diminution of tonus in the homolateral muscles, but bilateral destruction of the vestibular nuclei does not give rise to a complete disappearance of decerebrate rigidity. As a result of their experiments on decerebrated cats, these authors are led to believe that in addition to the vestibular nuclei, the large cells in the substantia reticularis, into which the fibers from the anterolateral columns are seen to enter, must be considered as playing an important rôle as centers of static innervation. The influence of the cerebellum on the tonus regulating mechanism, which after irritation of the lobus anterior finds expression in a removal of tonus distribution to the lower extremities in favor of the flexors, is not mediated solely through the brachium conjunctivae. There is a second efferent system which conducts cerebellar impulses to the medulla oblongata; this system is demonstrable in the restiform body, and is probably represented by the tractus fastigio-bulbaris. Another tonus regulating mechanism, besides that of the pyramidal tracts and frontal lobe, has its origin in the temporal lobe. Significant, however, as all these mechanisms for tonus may be, their influence in quadrupeds (carnivora) is slight.

KESCHNER, New York.

ACUTE ENCEPHALITIC PARKINSONISM. LEROY, J. Neurol. Psychiat. **25**:254 (April) 1925.

"It is generally admitted that the Parkinson Syndrome constitutes a late manifestation, a sequel, a complication of epidemic encephalitis." The author describes a case which followed acutely an attack of encephalitis. The patient was 30 years of age. About Jan. 1, 1925, the patient developed malaise and depression. His family physician thought he was suffering from a depression such as he had had about fifteen years before. On January 16 the author was called to see the patient, who at this time had a typical parkinsonian syndrome: the facies were fixed, the head slightly flexed, the attitude apathetic; he sat for half an hour without making the slightest movement. Objectively he showed moderate rigidity and hypertonus; the tendon reflexes were exaggerated in all four extremities, and there were no pathologic reflexes or pupillary findings. On January 20, the rigidity had increased, but the next day the patient was somewhat better. Lumbar puncture in March, 1925, showed a clear fluid, three cells, greatly increased pressure, and 70 mg. of sugar. The author has seen one other case of this sort with a typical encephalitic history.

There are only a few cases of acute postencephalitic parkinsonism recorded in the literature. Mlle. Levy observed it in a child, aged 1 year, about two and one-half months after the onset of the encephalitis. More recently Lemos reported the case of a young man, aged 18, who developed a Parkinson syndrome in the second week of encephalitis.

ALPERS, Philadelphia.

EXPERIMENTAL INTRODUCTION OF BIOLOGICALLY ACTIVE SUBSTANCES FROM THE CIRCULATION INTO THE CENTRAL NERVOUS SYSTEM. HANS HOFF and FRITZ SILBERSTEIN, Jahrb. f. Psychiat. u. Neurol. **43**:75, 1924.

This paper is devoted to a study of the experimental introduction of neo-arsphenamin from the circulation into the central nervous system. The authors found that they could introduce neo-arsphenamin into the central nervous system when the intravenous injection of arsphenamin was preceded by an endolumbar injection of sterile horse serum. The authors advocate the intravenous injection of neo-arsphenamin from eighteen to forty-eight hours after lumbar puncture; the arsenic will not reach the central nervous system if given ten or sixty hours after lumbar puncture. After the neo-arsphenamin has once entered the central nervous system by this method, relatively smaller doses injected repeatedly into the circulation and at shorter intervals will bring about an accumulation of arsenic within the central nervous system without the necessity for further lumbar puncture. The entrance of the arsenic into the nervous system seems to be accomplished by a sterile irritation of the meninges and choroid plexus. The irritation of these structures following the injection of the sterile horse serum is, at first, only of short duration, but seems to be prolonged after the arsenic has reached the spinal fluid. Flexner and Amoss obtained similar results by the injection of serum in cases of poliomyelitis. No ill effects were noted in any of the animals subjected to this experimental investigation.

KESCHNER, New York.

A MECHANISM FOR THE COORDINATION AND REGULATION OF CILIARY MOVEMENT AS REVEALED BY MICRODISSECTION AND CYTOLOGICAL STUDIES OF CILIATED CELLS OF MOLLUSKS. CASWELL GRAVE and FRANCIS O. SCHMIDT, J. Morphol. **40**:479 (Sept. 5) 1925.

Systems of fiber-like structures traverse ciliated cells and epithelia in a way to suggest conduction paths for stimuli for coordinating and regulating ciliary movement, and results of microdissection support such an interpretation. Some fibers of the systems in epithelia of the gill terminate on the nuclear membrane, some between nuclei in the basal cytoplasm. The observed double refringency of the fibrillar apparatus places it in the category of irritable and contractile substance. Bipolar cells and nervelike fibers lie immediately below and parallel with ciliated epithelia of the gill, but direct connections of these fibers with the intracellular system, although suspected, was not demonstrated. Ciliated epithelia of the gill are syncytial in character in the direction of the beat of cilia. Interciliary structures occur in the curricular portion of ciliated epithelia which are interpreted to function in taking up strains in the gelled cytoplasm caused by the beat of cilia. Cilia occur in pairs in epithelia of the gill, and members of the pairs are fused at their distal ends. Each cilium, as seen in dark-field illumination, pierces its basal body, and the fiber that is a continuation of the cilium divides immediately below the basal body into two—one diverging to the right, one to the left. Each cilium is composed of many delicate fibrillae.

WYMAN, Boston.

EXPERIMENTAL TUBERCULOSIS OF THE EAR. MASATO NAKAMURA, Arb. a. d. neurol. Inst. Wien. Univers. **27**:93 (May) 1925.

The experiments were conducted on guinea-pigs. Tubercle bacilli were introduced in one group of animals into the middle ear through the mastoid process, in another group through the tympanic membrane, and in a third group through the cervical portion of the common carotid. Prior to inoculation each animal was tested by means of Pryor's reaction and turning nystagmus tests to determine whether the labyrinth was intact. As a result of these investigations, Nakamura concludes that the disease does not extend to the brain until the labyrinth has become affected; extension to the brain from the middle ear without first involving the internal ear does not occur. In most cases involvement of the brain takes place through the internal auditory meatus by way of the lymphatic sheaths and, possibly, also through the apertura exterior aquaeducti vestibuli by way of the perilymphatic spaces to the subarachnoidea; and finally extension occurs by way of the blood channel. The process in the brain was either in the nature of a localized tuberculosis with a secondary extension to the meninges and the cerebrum, or in the nature of a meningoencephalitis. The process also involves the healthy ear through the perivascular spaces, but here the process is much less severe. All animals infected through the carotid artery developed a severe meningitis. It could not be determined whether the slight irritative process appearing simultaneously in both ears was the result of a vascular infection, or whether the meningitis gave rise secondarily to an infection of both ears.

KESCHNER, New York.

TREATMENT OF PARALYSIS BY MALARIA. T. W. DAVIDSON, Brit. M. J. **1**:452 (March 7) 1925.

Davidson here reports the results of the treatment in fifty-three cases of general paralysis with malaria during the year 1923. The infection was conveyed by mosquitoes in twenty-three cases and by inoculation (intravenous sixteen, intermuscular thirteen, subcutaneous one) in thirty cases. The incubation period varied with the route of infection, and averaged fourteen days in the mosquito infected cases, seven days in the intravenous, sixteen days in the intermuscular and twenty days in the subcutaneous cases. He reports the following results: Seven cases showed complete remission, nine cases showed both mental and physical improvement, seventeen cases showed slight mental with marked physical improvement. There were no mental nor physical changes in five cases, and there were fifteen deaths during the year — including six patients who collapsed under treatment. In the mosquito infected cases there were a few relapses of the malaria, but this rarely occurred in the inoculated cases. He concludes that malaria treatment of general paralysis is justifiable, especially in the early cases when the patient is in fair physical health.

POTTER, Akron, Ohio.

IODIDS AND THE CENTRAL NERVOUS SYSTEM. HANS HOFF, Arb. a. d. neurol. Inst. Wien. Univers. **27**:73 (May) 1925.

Hoff administered to fourteen dogs and twenty rabbits various preparations of the iodids. Some of the preparations were given by mouth, some intramuscularly and some intravenously. The animals were killed at various intervals, and the different organs and tissues were subjected to careful chemical examinations. Controls were also used. He found that in normal

animals alkaline iodids did not reach the central nervous system. In acute and chronic inflammatory processes, however, the simple alkaline iodids exerted their normal effect. In chronic degenerative processes lipiodin possibly had some effect, and may perhaps have some advantage over the alkaline preparations. Intravenous injection of iodids, in general, are advantageous because they reach the tissues more rapidly and are excreted more slowly. Pregl's solution (sodium hypoiodid) and colloidal solution of iodoform have no advantage over intravenous sodium iodid injections. The administration of thyroid preparations simultaneously with sodium iodid enables the latter to reach the central nervous system much more rapidly; this would seem the most desirable method of administering iodids in degenerative lesions.

KESCHNER, New York.

TREATMENT OF GENERAL PARESIS BY MALARIAL INOCULATION. P. BORREMAUS, J. Neurol. Psychiat. **25**:245 (April) 1925.

Since November, 1923, the author has treated by malarial inoculations thirty-seven patients who had general paresis. All the inoculations were made intravenously. Of thirty patients who received treatment for more than one year, ten were improved, and five of these left the asylum; fourteen were slightly improved or unchanged, and six died. At necropsy important cerebral lesions were found in the patients who died during treatment; in one there was a neo-membranous pachymeningitis with purulent areas and foci of calcification, while in another death was due to a meningeal hemorrhage. No cases of contagion were noticed during the course of treatment. The most frequent complication observed was jaundice, which occurred in four of more than thirty patients; in one case the jaundice was accompanied by an anasarca. The Bordet-Wassermann test which was taken before and after treatment showed little or no change in either the blood or the spinal fluid. On the contrary, the albumin content and lymphocytosis in the fluid was frequently diminished. The author considers the malarial treatment a favorable method of therapy, having had no success with the arsenicals.

ALPERS, Philadelphia.

EXPERIMENTAL INVESTIGATIONS ON MUSCLE TONUS. E. A. SPIEGEL, Jahrb. f. Psychiat. u. Neurol. **43**:165, 1925.

Spiegel conducted this investigation on seventeen animals (dogs and cats). He found that section of the anterolateral columns gave rise to a hypotonia of the homolateral limbs without interfering with ordinary voluntary movement. The hypotonia is attributed to a cutting off of the afferent proprioceptive impulses and partly also of the efferent extrapyramidal systems. So far as one can conclude from animal experimentation, section of the anterolateral columns will counteract hypertonia due to hyperexcitability of the proprioceptive tonus maintaining reflexes, such as one finds after a cutting off of the inhibitory centers in the brain stem following lesions in the pallidum. That this can be carried out in man without producing marked loss of function has recently been demonstrated by Frazier and Spiller when they sectioned these columns for the relief of incontrollable pain due to a malignant neoplasm of the spinal cord.

KESCHNER, New York.

THE MALARIAL TREATMENT OF GENERAL PARALYSIS. NORMAN B. GRAHAM, J. Ment. Sc. **71**:424 (July) 1925.

The experience of the Belfast Mental Hospital is given. The malarial parasites were brought from the Liverpool Tropical School of Medicine in

Mosquitoes which had fed on malarial paralytic patients at Whittingham Mental Hospital; these fed on four of the Irish patients and from the three who developed malaria of the simple tertian type, fifty-one cases were inoculated. The patient was allowed to have a series of rigors, determined by his physical condition, up to a maximum of twelve, with a course of quinin following. Of the first fifty patients, eighteen improved greatly, seven improved to the extent of being suitably employed, fifteen showed little or no improvement and ten died. Patients with early cases of the expansive and excited forms did best. One patient died in a seizure during the malarial fever.

Physical improvement was shown in gain of weight and loss of tremors, but not in changes of pupil or knee reflexes or in serology.

Of the ten deaths, probably five were influenced by the malaria.

The malarial treatment was not supplemented by any other method.

BOND, Philadelphia.

BILATERAL RECURRENT NERVE PARALYSIS DUE TO AORTIC ANEURISM. PAUL B. MACCREADY, Arch. Otolaryngol. **2**:122 (Aug.) 1925.

Both vocal cords were fixed in the position of adduction, causing marked dyspnea. A tracheotomy relieved this to some extent. A roentgenogram was then taken and an aneurysm noted, compressing the trachea and esophagus; this was confirmed by necropsy. Section of the right recurrent nerve showed a complete degeneration of all the fibers. Section of the left nerve distal to the aneurysm showed a small bundle with some fibers intact with the balance of the nerve degenerated. Neither nerve showed degeneration proximal to the aneurysm.

No satisfactory explanation has been found to account for the adduction in the case and in paralysis following operations for goiter. Experimental work has shown the abductor and adductor fibers as separate bundles in the dog, but this was not found in the present study.

HUNTER, Philadelphia.

ENCEPHALITIS LETHARGICA. C. M. H. HOWELL, Brit. M. J. **1**:437 (March 7) 1925.

Howell reports that since notification of cases of lethargic encephalitis were made compulsory in 1919, the following numbers of cases have been reported in London: 1919, 541; 1920, 890; 1921, 470; 1922, 454; 1923, 1123. The greatest number of cases was reported during the months of January, February and March in each year. This author reviews the etiology, symptomatology (quoting case reports to illustrate his symptom groups), sequelae, pathology and prognosis. Under differential diagnosis he calls attention to the fact that care must be taken to rule out cases of influenza and tuberculous meningitis, and states that the prognosis is often extremely difficult during the early course of disease. The article contains nothing especially new.

POTTER, Akron, Ohio.

BLOOD-SUGAR STUDIES IN MENTAL DISORDERS. S. A. MANN, J. Ment. Sc. **71**: 443 (July) 1925.

In establishing the blood-sugar curve in 152 cases of early mental disorder, the author is struck with the frequency of an abnormally sustained hyperglycemia following glucose ingestion. The suggestion is made that the efficacy of treatment by glands or drugs or diet may be indicated by the return of an abnormal curve to normal limits. Discussion of the effect of endocrins, acids, alkalis, pilocarpin and other drugs may be of interest to investigators.

BOND, Philadelphia.

Book Reviews

DER KRANKHEITSBEGRIFF IN DER KÖRPERMEDIZIN UND PSYCHIATRIE. VON DR. R. POPHAL, Privatdozent a. d. Univ. Greifswald, Nervenarzt in Stralsund. Price 5.70 marks, unbound. Pp. 111. S. Karger: Berlin, 1925.

The first half of this monograph is devoted to a critical review of the various concepts of disease in general from the days of Hippocrates to the present time. The most important concepts encountered in literature refer to the phenomenologic (not in a philosophic or psychologic sense), the morphologic or the etiologic point of view. The paramount question seems always to have been as to where, in the diseased organism, the locus morbi was to be placed. The answer given to this question may be said to be an index of the cultural attainment of the times and of the various stages in the evolution of medicine as a science. The most primitive disease concept encountered in the history of medicine is the mythologic, next the speculative-dogmatic, and finally, with the development of the natural sciences, the mechanic-materialistic concept. The last is probably best expressed by Virchow, who states that "the cell is not only the site of life but also the site of disease." All these concepts have in their content something in common that on final analysis can be crystallized into a principal content, which, according to Jaspers, is the relative biologic value that disease bears in the maintenance of the organism and its purposes. With this concept in mind, Pophal defines diseases as "changes in the course of life's processes, that, owing to a präexisting predisposition in the organism, can be brought about by certain noxa, which, injurious as they are in themselves, also become injurious to the individual affected; these changes may terminate in a complete cure, or in a cure with defects, or in death." The changes under consideration may be exclusively phenomenologic or both phenomenologic and morphologic.

The author then goes on to point out the urgent necessity of constructing disease entities so that one may formulate clear conceptions as to diagnosis, prognosis and ultimately, treatment. "Disease entities," he says, "are concepts whose content represents a sum total of all conditions bearing a common resemblance to each other." By including a given case in a known disease entity we can express in one or several terms the entire content of the general conception of that entity.

The next chapter is devoted to a discussion of the principles underlying a scientific classification of diseases. The existence of a greater number of syndromes than disease entities is given as the chief reason for the necessity of a systematic classification of combinations of pathologic manifestations not as yet looked on as disease entities. Several methods of classification have been in vogue from time immemorial; one of these is based on principles of pathologic anatomy, such as, atrophy, hypertrophy, necrosis, degeneration, etc.; another is that based on the principle of localization, such as gastric carcinoma, pulmonary embolism, etc.; and still another classification includes diseases characterized by the absence of a pathologic anatomy, such as infectious disease, blood and metabolic diseases, intoxications, etc. The last method, according to the author, leads only to confusion, because etiologic factors are placed on the same level as organic, phenomenologic and anatomopathologic factors. Classifying diseases on principles of heredity, constitution or on endogenous and exogenous factors is also faulty, because in the final analysis this

method is actually based on the principle of etiology. According to Pophal, all these methods are illogical and confusing, and in view of his definition of disease, he suggests the phenomenologic as the only logical principle for scientific classification; however, as pathologic-anatomic changes are after all merely manifestations of disease, one could perhaps construct a classification with special emphasis on a distinction between diseases with and diseases without pathologic-anatomic changes. Such a classification would have to be based on one principle only, and any other principles that would have to be taken into consideration in the classification would of necessity assume a subordinate and not a coordinate rôle. The practical application of some such method of classification would be a problem for every branch of medicine to solve for itself.

The second half of the monograph is devoted to a discussion of the nature of disease concepts in psychiatry. The discussion is practically along the same lines as that employed in somatic medicine. The book concludes with a historic sketch of the struggle which has been going on for centuries between the humorists and the solidarists as to whether the fundamental basis of mental disease is to be sought in the psyche or in the soma. This struggle apparently terminated in 1904 with Ziehen's adoption of the principles of neuropathology (somatic), to which he added experimental psychologic methods of investigation. According to Pophal, this apparently most logical approach to the problem is now being threatened by the "phantastic" psychanalysts with their "one-sided and far-fetched interpretations" of mental phenomena. In closing this part of the monograph the author sounds a timely note of warning against the recent tendency by many psychiatrists to look at mental disease from a purely psychologic point of view.

An appended bibliography containing 220 references to as many quotations in the text attests that the author must have read widely and well, and that he has a real grasp on the problems discussed. While it cannot be said that the book is practical, nevertheless the reviewer believes that it can be read to advantage by all neuropsychiatrists, and it should be read by all physicians who in the course of their routine work see in disease something more than a combination of signs and symptoms needing treatment.

TEXT-BOOK OF NERVOUS DISEASES. FOR THE USE OF STUDENTS AND PRACTITIONERS OF MEDICINE. By CHARLES L. DANA, A.M., M.D., LL.D., Professor of Nervous Diseases in Cornell University Medical College; Consulting Physician to Bellevue Hospital; Neurologist to the Montefiore Hospital; Consultant and Trustee of the New York Neurological Hospital; Consulting Physician to the Manhattan State Hospital; Ex-President of the American Neurological Association; Ex-President of the New York Academy of Medicine; Corresponding Member of the Société de Neurologie, etc. Tenth Edition. Price, $7.00. Pp. 667, with Two Hundred and Sixty-Two Illustrations Including Four Plates in Black and Color. New York: William Wood & Company.

For about thirty-three years this textbook has been one of the very best one volume works on diseases of the nervous system. The present edition has not only been fully revised and brought up to date, but contains some new and attractive features. Dr. Fielding H. Garrison, "by far the highest authority on medical history in this country," has contributed a chapter of forty pages on the history of neurology which, we believe, could not be improved. It not only contains an enormous amount of information, but is pleasingly written. It is a very valuable historical document.

Another innovation is a short chapter on preventive neurology, which should help neurologists and other physicians to take a broader view of their duties and of the function of medicine.

The chapter on lethargic encephalitis has been rewritten, and is good.

In a work of this size covering an enormous subject, the problem of what to include and what to reject is never solved to the satisfaction of all, probably rarely to the satisfaction of the author. In the present instance it appears to us that the author's well-known calm judgment and unbiased attitude are well evidenced in the arrangement of his textbook. The work is compact, devoid of verbiage, devoted to essentials and is peculiarly well balanced; a safe guide which really presents quite fairly neurology as it is today.

The figures are illustrative, the tables helpful, the index adequate and the make-up good.

GRUNDZÜGE DER ÄRZTLICHEN PSYCHOLOGIE, PSYCHODIAGNOSTIK UND PSYCHOTHERAPIE IN DER TÄGLICHEN PRAXIS. DR. HEINZ FENDEL. Price, 3 marks. Pp. 134. Berin, Urban & Schwarzenberg, 1925.

Fendel addresses his book to the general practitioner and tries to give a summary of recent views in the field of medical psychology. He discusses the various phases of psychic experience and presents present concepts of the unconscious, inhibitions, sexuality, symbolism, uncritical attitudes, repressions, suggestion, psychogenesis, etc., as held by various authors of the day.

He also discusses the particular histories necessary for psychodiagnosis. The chapters on therapy include psychanalysis, suggestion, hypnosis, psychogogie, or the teaching of inhibition through systematized practice, and logotherapie, or consistent and logical reasoning by the physician with the patient regarding his ailment.

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